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ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

THE APPEARANCE AND BEHAVIOR OF THE NORMAL TYMPANIC MEMBRANE.

DR. GEORGE W. MACKENZIE, Philadelphia, Pa.

The subject of the paper was suggested to the writer for what appeared to him very good reasons:

1. That the average text-book does not inform the student sufficiently concerning the anatomy, the otoscopic appearance and the behavior of the normal tympanic membrane; and unless they are understood by the student otologist he is handicapped from the very start.

2. That careful observations, both anatomical and with the magnifying Siegle otoscope, have led the writer to certain information concerning the appearance of the tympanic membrane and its behavior, which he feels are worth while publishing for the benefit of those who have not had the time or opportunity to ascertain the same information for themselves.

3. That since the writer has presented the subject before informally, and then only in a fragmentary way, and not for publication, he feels that the time has arrived for him to present the subject more comprehensibly and with the object of establishing priority concerning certain of the findings and interpretations, which are original with him.

The first thing to take up in the consideration of a subject of this kind is the anatomy of the parts. The best and most recent authoritative work on the subject of the anatomy of the tympanic membrane is that of Schwalbe.[1] He devotes twenty-two pages to the consideration of the anatomy of the drum head and makes use

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of references from fifty-seven authorities. The present writer took the time to run these out, as far as possible. Unfortunately about one-third of the references were not available in this country, so that in some instances recourse was limited to second-hand information, which is never so reliable as first-hand. On the whole, the literature was excellent, especially that which has appeared in the English language, including the works of Home,[2] Shrapnell,[3] Toynbee,[4] Wharton Jones,[5] Burnett, [6] Crombic[7] and Howes.[8]

The intention, on this occasion, is not to present the anatomy of the tympanic membrane as detailed as might be done; but rather to present those most salient points in the anatomy which bear directly on the subject matter of the paper.

With the liberal use of illustrations the writer hopes to reduce as much as possible word pictures, which tend to strain the attention of the listener or reader. However, word pictures are not to be entirely ignoerd.

The drum head—Membrana tympani—separates the external auditory canal from the drum cavity. The larger part (about ⅚) of the circumference of the drum head is inserted into the "U" shaped Sulcus tympanicus of the tympanic bone. The anterior superior extremity of the annulus, which carries the sulcus, is known as the Spina tympanica anterior of Henle. The posterior superior extremity is known as the Spina tympanica posterior. The posterior spine is situated at an appreciably higher level than the anterior. The gap in the tympanic bone between these two spines, occupied by the horizontal plate of the squamous bone, is known as the Incisura Rivini. The area between the two spines just mentioned and between the short process of the hammer below and the Margo tympanicus of the squamous bone, is occupied normally by the Membrana flaccida, first described by Shrapnell and named after him. The remainder of the drum head is known as the Pars tensa.

The boundary which separates the Pars flaccida and Pars tensa is defined by two folds, a short (1½ m.m.) anterior fold, which reaches from the short process of the hammer to the anterior tympanic spine, and a long (3½ m.m.) posterior fold, which extends from the short process of the hammer to the posterior tympanic spine. The prominence of the short process causes these folds to extend somewhat above (externalward) the level of the rest of the tympanic membrane. These folds, together with the other descriptive matter up to this point, is best shown in the illustration taken from Schwalbe's Fig. 165 which appears as Fig. 1 in this paper. Directly on the inner surface corresponding to the anterior

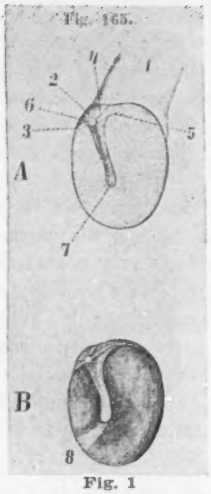

Fig. 1

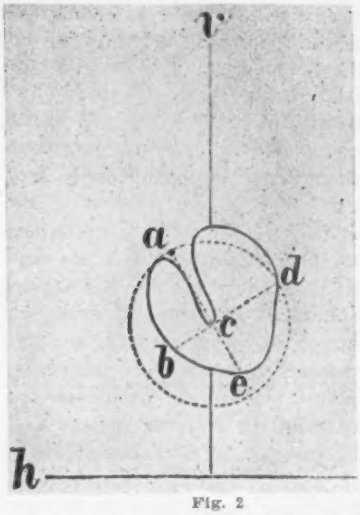

Fig. 2

Fig. 1. Taken from Dr. G. Schwalbe, 1887, Lehrbuch der Anatomie des ohres, page 441, fig. 165. **A**, Left drum head. View from external canal side; anterior margin of drum head is to the left, posterior margin to the right. 1, Superior wall of external canal; 2, Membrana flaccida; 3, anterior fold of the drum head; 4, superior fold of the drum head; 5, posterior fold of the drum head; 6, tip of the short process; 7, umbo; between 6 and 7 Stria malleolaris.

B, Otoscopic view of drum head (from Politzer). 8, Light reflex. The three folds (3, 4, 5) as seen in figure A are exaggerated because it is taken from a wood cut.

Fig. 2. Taken from Dr. G. Schwalbe, 1887. Lehhbuch der Anatomie des Ohres, page 444, fig. 166. Horizontal projection of the outer surface of the left drum head. ac, Stria malleolaris. With the radius distance from point a to point c a circle circumscribed about the point c is represented by dots, it shows just how much the circumference of the drum head deviates from a circle. abc, anterior superior quadrant; bce, anterior inferior quadrant; dce, posterior inferior quadrant; acd, posterior superior quadrant.

fold lies the Ligamentum mallei anterius, which is grasped so to speak by the Sulcus malleolaris, which lies directly below the Crista spinarum. The posterior fold, according to Schwalbe, spreads out into and blends with the beginning of the posterior leg of the Annulus fibrosus, which occupies the Sulcus tympanicus.

The most prominent portion of the short process of the hammer corresponding to the most elevated position of these folds, is known as the Prominentia malleolaris. Extending from the Prominentia malleolaris, along the middle of the hammer handle and lengthwise with it clear to the spatulated end at the umbo, is a line of attachment for the drum membrane to the manubrium, known by some authors as the Stria malleolaris. Some authors do not recognize the Stria at all. According to Schwalbe it is a line which marks the firmest attachment of the membrane to the hammer. It is illustrated in Fig. 1,

taken from Schwalbe, extending from points 6, Prominentia malleolaris to 7, the spatula. The line of the anterior and posterior folds lies at right angles to the Stria malleolaris. The writer, too, has observed a line corresponding to Schwalbe's Stria malleolaris; but it is not exactly the same as the one described by him; at least the writer sees it differently. The line as observed by the writer divides the hammer handle longitudinally into unequal halves, the anterior corresponds to that portion of the hammer handle which is in intimate contact with the drum head, where it is impossible to get anything between them; while posteriorly to the line the hammer handle is not in contact with the drum head, since it is possible by aspiration with the Siegle otoscope (negative pressure) to lift this part of the membrane clear away from the hammer handle. When compression is used the drum membrane overlying the posterior half of the hammer handle can be made to approach the lateral aspect of the posterior half of the hammer handle, but under no circumstance can that part of the drum membrane which lies over the posterior half of the hammer handle and which is normally not in intimate contact with the hammer handle be made to appear as though it was, or as it appears in the case of the anterior half.

There is another fold which extends from the Processus brevis of the hammer in an upward and slightly backward direction, known as the superior fold. This superior fold divides the space occupied by Schrapnell's membrane into two triangles. The angle formed by the anterior and the superior folds, which meet at the Prominentia malleolaris is quite obtuse (exceeds 130 degrees); while the posterior angle formed by the posterior and superior folds meeting at the Prominentia malleolaris is less than 50 degrees.

That part of the Membrana flaccida lying anterior to the superior fold comprises a larger field than that lying posterior to the superior fold. The posterior or smaller field is normally *not* sunken or retracted like the anterior and is more tense. The anterior field is quite flaccid and slightly depressed; however, by Politzer inflation this relaxed depressed flaccid anterior field will reverse its position and come outward and remain so for a while, when it will under normal conditions of the Eustachian tube and middle ear, very gradually resume its primary position. The posterior or smaller field of the Membrana flaccida is covered with a strip of modified skin, extending downward from the superior wall of the canal. The thickest or densest part of the strip corresponds to the superior fold. It is this strip of skin that carries the superficial vessels (arteries and veins) and the nerve of the membrane, all of which parallel the long axis of the hammer handle, but always *pos-*

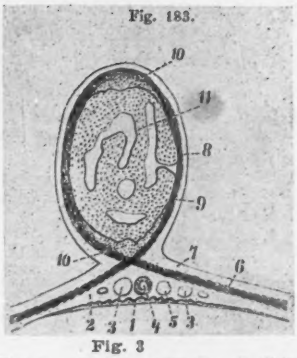

Fig. 3

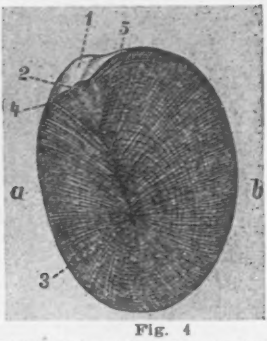

Fig. 4

Fig. 3. Taken from Dr. G. Schwalbe, 1887. Lehrenbuch der Anatomie des Ohres, page 500, fig. 183. Cross-section through the Manubrium mallei in connection with the drum head. The cut is perpendicular to the long axis of the hammer about the middle between the Processus brevis and the umbo. The skin layer of the drum head shows here in the cross section the cuticular stripe with its thickened epithelium. 1, its dense connective tissue base; 2, the veins; 3, the artery; 4, and the membranae tympani nerve; 5, the connective tissue sends low papilla into the epithelium; 6, radial fibre layer of the drum head, obliquely cut, crossing over into the periosteum of themanubrium; 7, mucous membrane layer of the drum head; 8, mucous membrane layer of the hammer handle; 9, periosteum of the hammer handle; 10, remnant of cartilage; 11, bone.

Fig. 4. Taken from Dr. G. Schwalbe, 1887. Lehrbuch der Anatomie des Ohres, page 456, fig. 169. Left drum head. Schematic representation of the fibres of the Membrana propria. a, anterior; b, posterior margin of the drum head; 1, Membrana flaccida; 2, Processus brevis of the hammer showing through; 3, umbo of the drum head; between 2 and 3 is shown the lateral edge of the hammer, at which the radial fibres unite with the periosteum of the hammer handle; 4, anterior superior end of the Sulcus tympanicus; 5, posterior superior end of the same; from here the fibres go to the Processus brevis.

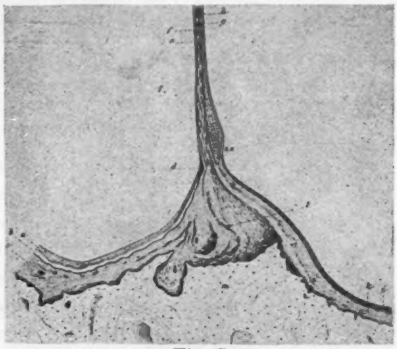

Fig. 5

Fig. 5. Taken from Gustav Brunner, 1870. "Beitrage zur Anatomie und Histologie des mittleren Ohres", tafel 1, fig. 1. Radical section through the lower margin of the drumhead. To the left is the external auditory canal. To the right is the floor of the drum cavity; a, epidermis of the external canal; b, malpighian layer; c, corium with evidence of papillae and subcutaneous connective tissue; d, thickened epidermis where the canal and drum membrane meet. Drumhead; e, external dermal layer; f, radiating fibre layer; g, circular fibre layer; h, mucous membrane of the drum cavity; i, moist epithelium, at xx location where the transition of epithelium occurs; k, connective tissue stratum with its own vessels; l, Annulus tenderness (Ringwulch); m, Sulcus tympanicus of the temporal bone.

terior to the Stria malleolaris. That the anterior field of Shrapnell's membrane is thinner than the posterior can be proved when one inflates the tympanic cavity with coloring matter, for instance Berlin blue, it will be found that the coloration shows through the thinner anterior field quite distinctly, but not at all through the thicker posterior field. Neither the posterior nor the anterior field contains a Substantia propria as is found in the more tense portion of the membrane; in other words, that portion which lies below the anterior and posterior folds and which finds attachment in the Sulcus tympanicus.

Referring to Shrapnell's membrane, Crombie,[7] in criticising Helmholtz[9] treatise on the mechanism of the ossicles, claims that an explanation of the mechanism of the Membrana tympani which takes no account of the Membrana flaccida (its upper portion), can at best, even if otherwise correct, be but incomplete and defective.

The outline of the Membrana tensa is less of a circle than it is an irregular ellipse in shape. If an imaginary line should be extended downward and slightly backward through the Stria malleolaris to the periphery of the membrane, it will divide the surface of the tense portion of the drum membrane into two slightly unequal sized fields—an anterior one and a posterior. The posterior field is larger than the anterior. A second line drawn at right angles to the first and through the umbo divides the drum head into a superior and an inferior field. The superior is slightly larger than the inferior, see Fig. 2 taken from Schwalbe's Fig. 166; also sketches by the writer, Figs. 27 and 28. The primary axis, drawn through the Stria malleolaris, is inclined downward and backward making a 30° angle with the vertical. The length of the primary axis, measured from the Prominentia malleolaris to the opposite rim, averages about 9.75 m.m. This does not correspond to the longest dimension of the ellipse; that is, the distance from the point where the upper vertical line v in Fig. 2 touches the upper margin of the drum membrane down to the point e on the inferior margin. This diameter equals approximately 11 m.m. The breadth of the membrane measured along the secondary axis equals about 9 m.m., while the greatest breadth which is at right angles to the longest dimension, equals about 9.5 m.m.

The above figures are the average taken from the measurements presented by Gruber,[10] Politzer,[11] Kessel[12] and Tröltsch.[13]

The membrana tensa, as its name implies, is more tense than the flaccid portion. The reason for the tensity of the tense portion as compared with the flaccid is that in the former we find a fibrous

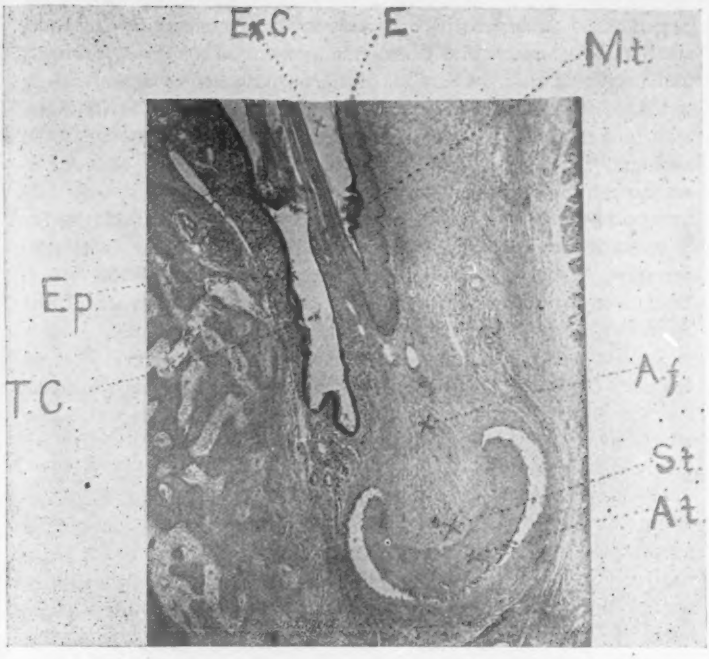

Fig. 6. (Author.) e. epidermis of the external canal; ExC, external auditory canal; Mt, Membrana tympani; Ep. epithelial lining of the tympanic cavity; At, Annulus tympanicus; St, Sulcus tympanicus; A, Annulus fibrosus.

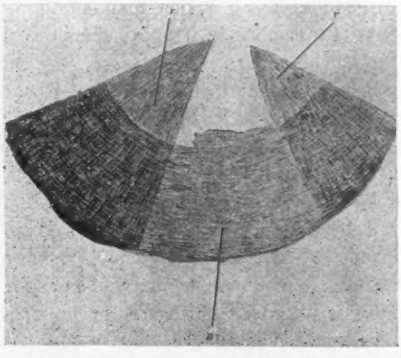

Fig. 7. Taken from Prof. Dr. G. Alexander, 1912. "Die Ohrenkrankheiten im Kindesalter", page 20, fig. 26. Radial (r) and circular (z) fibres of the drum head.

connective tissue stroma, Substantia propria, which is absent in the latter. The Substantia propria adds to the thickness and resistance of the Pars tensa. The Substantia propria is composed of many radiating and circular bundles of fibrous connective tissue, closely arranged. This tissue is particularly thick and strong at its insertion in the groove or sulcus of the Annulus tympanicus, where on account of its location and character, it is frequently referred to as the Annulus fibrosus. It is an important structure, not only from a physiologic point of view, but also from the pathologic, for it is the last to disappear in the melting away of tissue from suppuration. The whitish opaque appearing margin of the drum head, seen on otoscopic examination, indicates the presence of the Annulus fibrosus. On the outer surface of the Substantia propria is the Stratum cutaneum, a modified layer of the external skin of the canal. On the opposite side of the drum head lining the inner surface of the Substantia propria, we find the Stratum mucosum, which is merely the extension of the mucous membrane lining of the tympanic cavity.

The skin layer—Stratum cutaneum—Dermoid layer of Toynbee[4] is, on the average, quite thin (50-60μ) but in the region of the manubrium it is thick and strong (0.5 m.m.). This thicker, more resistant, strip of skin which extends downward from the upper wall of the canal, the bande cutanée of Tillaux,[14] follows just behind the Stria malleolaris to the umbo. Arnold, quoted by Schwalbe, had earlier believed this band a ligament and designated it the Ligamentum mallei externum. Casserius, on the other hand, believed it to be a muscle and called it Musculus tympani minor S mallei externus. At the upper margin of the drum membrane this band or strip of skin is much wider than along the manubrium and narrows down appreciably as it approaches the spatulated end at the umbo.

According to Schwalbe the thickness of the Stratum cutaneum of the more transparent part of the drumhead is only 16-20μ, while the thickness of the bande cutanée of Tillaux or Cutisstranges of the Germans, just behind the Stria malleolaris is 320μ. This thicker cuticular layer contains papillae, the principal vessels of the drum head (large artery and two veins) and a large nerve. (N. membrana tympani.) See Fig. 3, which is a reproduction of Schwalbe's Fig. 183. It would seem that the purpose of this strip of thickened cutis is to support and carry the important structures bound up in it.

Substantia propria, Membrana propria, Membrana fibrosa, the fibrous middle layer of the membrane is formed of two flat layers

of smooth dense connective tissue fibres arranged in compact bundles. According to their arrangement one can divide them into two distinct lamella. The fibres of the outer layer are arranged radially and run a straight course. Fibrae rectae of Moldenhauer.[15] They form a complete membranous disk, the periphery of which forms a thick, tough, opaque, circular, fibrous ring which is inserted into the Sulcus tympanicus on the one hand and the hammer handle on the other (Tröltsch). The inner layer of connective tissue fibres is arranged more or less in a circular fashion and is known as the circular fibre layer. Both these layers are shown to advantage in Fig. 4 taken from Schwalbe's Fig. 169.

Fig. 19.

Fig. 8

Fig. 9

Fig. 8. Taken from Politzer. Lehrbuch der Ohrenheilkunde, 1908, page 16, fig. 19. Segment of the lower part of the drum head; h, hammer handle; r, radial fibre layer (Stratum radiatum); c, circular fibre layer (Stratum circulare).

Fig. 9. Taken from Prof. Dr. G. Alexander, 1912. "Die ohrenkrankheiten im Kindesalter", page 22, fig. 29. Dendritic network (D) on the median surface of the drum head (MT).

The Annulus fibrosus or tendinosus is found in the periphery only as far as the Sulcus tympanicus extends. The Annulus presents the appearance, upon otoscopic examination, of a narrow, white, opaque zone at the periphery of the Pars tensa. More white and opaque than the rest of the Pars tensa with the exception of the more or less opaque zone about the spatulated end of the manubrium. This latter presents really less opaqueness and whiteness otoscopically than the visible part of the Annulus fibrosus. To facilitate a firmer union between the Annulus fibrosus and the Sulcus tympanicus the bone presents a jagged raw edge, see Fig. 5.

Fig. 10

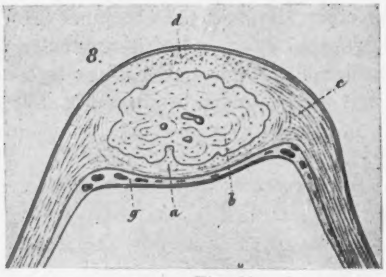

Fig. 11

Fig. 10. Taken from Dr. G. Schwalbe. Lehrbuch der Anatomie des Ohres, 1887, page 500, fig. 182. Cross-section through the lower spatulated end of the hammer handle in connection with the drum head. 1, epithelium; 2, connective tissue of the skin layer; 3, periosteum of the hammer handle; 4, cartilaginous covering of the hammer handle; 5, bone; 6, radial fibre layer of the drum head passing over into the perichondrium; 7, mucous membrane.

Fig. 183. Cross-section through the Manubrium mallei in connection with the drum head. The cut is perependicular to the long axis of the hammer about midway between the Processus brevis and the umbo. The skin layer of the drum head shows here in cross section the cuticular stripe with its thickened epithelium: 1, its dense connective tissue base; 2, the veins; 3, the artery; 4, and the Membrana tympani nerve; 5, connective tissue sends low papilla into the epithelium; 6, radial fibre layer of the drum head, obliquely cut, crossing over into the periosteum of the manubrium; 7, mucous membrane layer of the drum head; 8, mucous membrane of the hammer handle; 9, periosteum of the hammer handle; 10, remnant of cartilage; 11, bone.

Fig. 184. Cross-section through the drum head in connection with the short process of the hammer; 1, epithelium of the cutaneous layer of the drum head; 2, cuticular layer of the drum head; 3, Membrane propria; 4, perichondrium of the short process of the hammer; 5, mucous membrane of the drum head; 6, mucous mebrane of the hammer; 7, cartilage of the short process; 8, bone with large blood vessel spaces; 9, fibrous foundation of the posterior pocket bands.

Fig. 11. Taken from Gustav Brunner, 1870. "Beitrage zur Anatomie und Histologie des mittleren Ohres", tafel 2, fig. 8. Cross-section through the hammer handle of an adult perpendicular to the same and to the surface of the drum membrane at the spatulated end. The lateral aspect of the drum head is directed downward: a, cartilage; b, bone with Haversian canals and the lamellated system; c, fibres of the drum head; d, fibres of the drum head in cross-section; g, dermal layer of the drum head with blood vessels.

reproduced from Brunner's Fig. 1, and compare with photographic illustration, Fig. 6, taken from the writer's collection, which does not show the sagittations, at least not to the same degree as illustrated by Brunner.

The radial fibre layer (Stratum radiatum) is formed of very fine (4-10μ thick) flat bundles of connective tissue. The bundles, generally, appear homogenous and stiff; however, after treating the

Fig. 12

Fig. 13

Fig. 12. Taken from Gustav Brunner, 1870. "Beitrage zur Anatomie Histologie des mittleren Ohres, tafel 2, fig. 11. Cross-section through hammer handle of an adult, perpendicular to the same and perpendicular to the surface of the drum head. The lateral surface of the drum head is directed downward in the illustration. Anterior direction is towards the right. This section is made a short distance below the level of the short process: a, cartilage; b, bone with Haversian canals and lamellated system; c, connective tissue fibres of the drum head; 1, Chorda tympani nerve N. B. In a triangular space, corresponding to Brunner's posterior discontinuat he has placed a rosette-like figure representing whirls of connective tissue fibres. Compare this figure with Brunner's figure 13 which is taken at a lower level where he has represented at e, posterior discontinuat, an open space. See further reference to this space in the text.

Fig. 13. Taken from Gustav Brunner, 1870. "Beitrage zur Anatomie und Histologie des mittleren Ohres", tafel 2, fig. 13. Cross-section through the hammer handle which is also perpendicular to the surface of the drum head. The lateral aspect is below and the anterior direction is to the right. This section is through the lower third of the hammer handle but above the spatulated end: a, cartilage; b, bone; c, connective tissue fibres of the drum head; d, connective tissue fibres in cross-section; e, posterior discontinuat of Brunner; f, anterior discontinuat of Brunner; g, dermal layer of the drum head with blood vessels. Note the difference in the thickness of the skin layer as represented by Brunner in this figure and that of Schwalbe in figure 3.

drum head with chromic acid and sodium chlorid solution 10 per cent, one can readily discern the fibrillary arrangement of these bundles. Besides the resistance of the fibres to the action of acids and alkalies is the same as occurs in the case of fibrillary connective tissue fibres anywhere else. These stiff connective tissue bundles lie in strata or lamella, which divide and again reunite, making very acute angles directed away from the centre of the drum head. The general course of the fibres are radial. The direction of the intervening spaces between the fibres is also radial. The radiation of the fibres is not from a common centre at the umbo except for those which are situated in the inferior half of

the drumhead. For the radial fibres of the superior half the center of radiation is a modified centre, that is a line represented by the length of the Stria malleolaris, see Fig. 4 after Schwalbe, also Fig. 27, after the writer. In other words, those radiating fibres situated above the level of the umbo, that is of the superior posterior and superior anterior quadrants, radiate decidedly less than do the fibres of the two inferior quadrants. Above the anterior and posterior folds which mark the separation of the Membrana flaccida and Membrana tensa the radiating fibres cease to exist. Between the radial running fibres there is a system of radial running lymph spaces, lining the walls of which are to be distinguished flat endothelial cells. Formerly these endothelial cells were believed to have been a particular form of corpuscle which Tröltsch designated Trommelfell Körperchen. So far as the fibre bundles, cells and lymph spaces are concerned and their anatomical arrangement, the drumhead is comparable in structure with the cornea of the eye.

The circular fibre layer of the Membrana propria structurally resembles the radial, in that it is composed of homogenous appearing bundles of fibrous connective tissue. As the name indicates, the circular fibres run in a more or less circular direction and at right angles to the radial fibres. Besides the circular fibres are generally finer than the radial and somewhat elastic, so that after separation they contract (Toynbee). The circular fibre layer begins at the tendinous ring (Annulus tendinosus) on the inner surface of the radial fibre layer where it presents its greatest thickness (Fig. 5), which is frequently twice as heavy as that of the radial layer. The circular layer diminishes in thickness from the periphery toward the hammer handle. The peripheral third of the circular layer is composed of several thicknesses. Midway between the Annulus and the Manubrium the thickness is reduced to about a single layer, while at the spatulated end the circular fibres are barely discernible. See Figs. 5, from Brunner; 7 from Alexander,[21] and 8 from Politzer.

The union of these two (circular and radial) fibre layers of the Membrana propria is not very firm. They are united by a loose meshwork of very fine connective tissue fibres which run in different directions.

According to Kessel the delicate outer cuticular layer is quite loosely attached to the radial layer by connective tissue fibres. The two are held together by a loose network of fibres similar to that which holds the two fibrous layers together. The union between the Substantia propria and the mucous membrane in the anterior and inferior part of the Pars tensa is quite the same as

between the Substantia propria and the cutis. In the posterior and superior parts both the circular and the radiating fibres take part in the union, forming trabecular criss-crossing in all directions, making a stonger union than otherwise might occur. Gruber, who first described this union, referred to it as dendritic fibre formation of the drumhead. Some of the fibres reach from the drumhead in different directions into the drum cavity itself. Alexander quotes Gruber as authority for the belief that the mucus layer of the drum membrane in conjunction with the Lamina propria, forms folds and trabecular thickening, which, in a flat aspect, appear as a dendritically ramified network, see Alexander's Fig. 29, which is reproduced here as Fig. 9. The Membrana propria stands in intimate relation with the manubrium, more about which shall be said later.

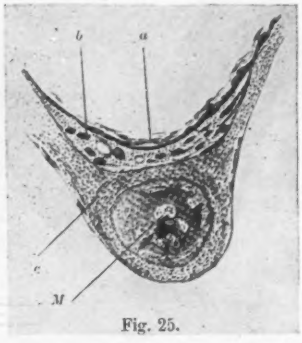

Fig. 25.

Fig. 14. Taken from Prof. Dr. G. Alexander, 1912. "Die Ohrenkrankheiten im Kindesalter", page 19, fig. 25. Cross-section through the drum head of a new born at the level of the hammer handle: M, manubrium mallei; c, circular connective tissue fibres surrounding the manubrium; b, vessels which follow the hammer handle; a, epidermal layer with desquamation of the cells.

The mucous membrane layer of the drumhead (Stratum mucosum) covers the Membrana propria on its median or tympanic surface. It is like the mucous membrane of the tympanic cavity of which it is a continuation. The Stratum mucosum of the drumhead consists of surface epithelia and thin subepithelial connective tissue. The epithelial surface is composed of a simple layer of cuboid cells without cilia. The connective tissue of the mucous membrane forms a delicate network, among which are to be found some elastic fibres. This connective tissue tends to follow the dendritic fibre arrangement of Gruber, referred to earlier.

The manner of the attachment of the hammer to the drum membrane is a very important consideration from many aspects as will

be pointed out further on. The following description is that which is commonly accepted as the normal anatomical relationship. See Fig. 10, taken from Schwalbe's Figs. 182, 183, 184; Figs, 11, 12, 13, taken from Brunner's Figs. 8, 11 and 13; and Fig. 14, taken from Alexander's Fig. 25.

According to Schwalbe, who summarized the works of many investigators, the attachment of the hammer to the drumhead occurs along the whole lateral edge of the hammer handle from the Porcessus brevis to the spatula. See Fig. 10. The firmest attachment is at the spatula and the second firmest attachment is at the Proces-

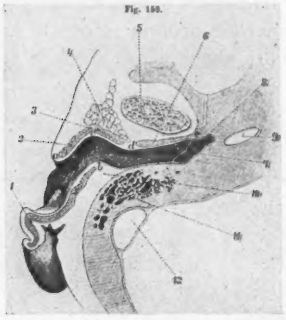

Fig. 15

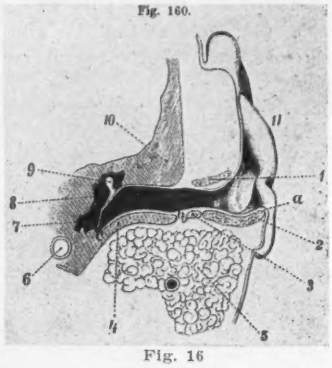

Fig. 16

Fig. 15. Taken from Dr. G. Schwalbe, 1887. Lehrenbuch der Anatomie des Ohres, page 425, fig. 159. Horizontal section through the external auditory canal. Bone=lines; cartilage=dots; lumen of the external auditory canal is dark. 1, cartilage of the external ear; 2, tragus; 3, median part of the anterior wall of the cartilaginous canal; 4, parotid gland; 5, head of the condyle of the lower jaw; 6, Os tympanicum, anterior wall of the bony canal; 7, drum head; 8, tympanic cavity; 9, internal carotid; 10, fibrous part of the posterior wall of the canal; 11, mastoid cells; 12, transverse sinus.

Fig. 16. Taken from Dr. G. Schwalbe. Lehrbuch der Anatomie des Ohres, 1887, page 426, fig. 160. Vertical cut through the external auditory canal. Bone=lines, cartilage=dots; ab, cartilaginous; bc, bony canal; 1, curled plate of the tragus in the upper canal wall; 2 and 3, floor of the cartilaginous canal; between 2 and 3 incusura santorini minor; 4, Os tympanicum; b, connective tissue between the cartilaginous and bony canal; 5, parotid gland; 6, internal carotid; 7, drum head; 8, tympanic cavity; 9, hammer; 10, Os squamosum; 11, auricle.

sus brevis; while along the hammer handle between these two places the union is merely, by the narrow edge of the hammer to the membrane. See Fig. 3. In the region of the spatulated end of the Manubrium a cross-section shows it to be somewhat elliptical shaped at the Superficies umbilicalis. See Figs. 10 and 11. The long axis of this ellipse is parallel to the surface of the membrane; while the cross-section of that part of the Manubrium between the spatulated end below, and the short process above, presents also an ellipse, but in this latter case the long axis of the ellipse is at

right angles to the surface of the membrane, according to Schwalbe, see Fig. 3. At the Processus brevis the very tip is somewhat flattened and covered with cartilage, thereby affording a relatively broad surface for the attachment of the drum membrane. The materials which bind the hammer handle to the membrane is the periosteum or perichondrium on the one side and the Membrana

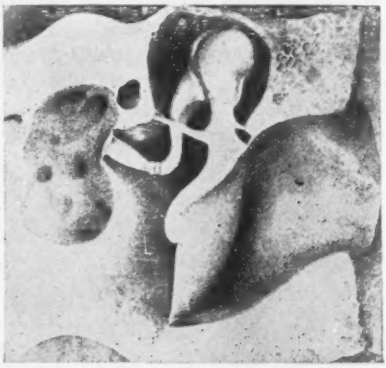

Fig. 17. Taken from Politzer's Atlas. Showing the relationship of the external osseous canal and the drum head to one another, also the ossicles and their relative position to one another. Note the normal concavity of the drum head.

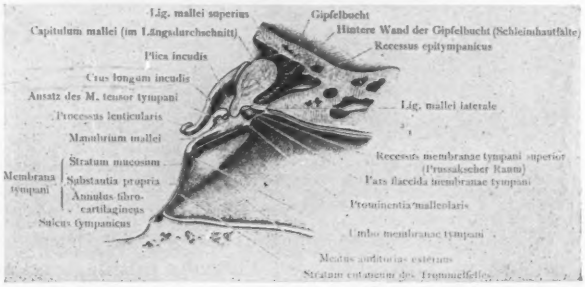

Fig. 18. Taken from Toldt. Frontal section through the left drum membrane and its bordering structures, including the external osseous canal and part of the middle ear cavity and its contents. Page 925, fig. 1423.

propria of the drumhead on the other. Fibrous bundles of the latter continue over into the fibres of the periosteum of the hammer handle. Fig. 3. The mucous membrane layer of the drumhead completes and secures the union.

Th blood vessels of the drum membrane arise from the external auditory canal on the one side and the mucous membrane lining of the tympanic cavity on the other. Communications between these

two systems occur in the neighborhood of the hammer handle and at the periphery of the drum membrane. According to Wendt[16] and Kessel there exists also a capillary network in the Substantia propria which is in communication with the vessels of the external and internal surfaces. Moos[17] does not accept this view of Wendt and Kessel. He found, on the contrary, rich communications between vessels of the skin and those of the mucous membrane by way of perforating veins in the intermediate area of the drumhead, that is, in the region between the Annulus tympanicus and the Manubrium mallei. According to Popper[18] the Membrana propria possesses no blood vessels with the exception of the peripheral part where the anastomoses between the vascular networks of the skin and mucous membrane takes place.

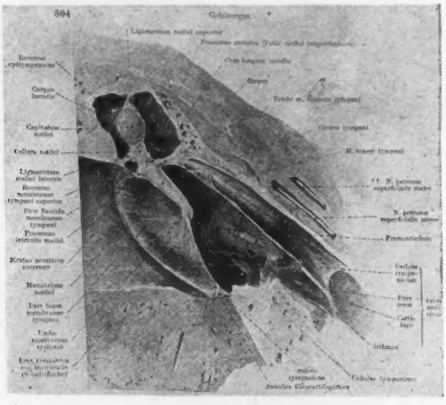

Fig. 19. Taken from Spalteholz. Handatlas der Anatomie des Menschen. Band 3, 1903, page 804, fig. 884. Vertical cut of the right drum head.

The anatomical researches of Prussak[19] on the drumhead of dogs, led him to believe that the Substantia propria contains no blood vessels. The cuticular layer (Stratum cutaneum) receives arterial blood through numerous small arteries which come from the skin of the canal. These invade the membrane from all directions. They soon break up into capillaries which run a converging course toward the center of the drumhead. Another source of blood supply for the Stratum cutaneum is from the Arteria auricularis profunda which gives off a branch which courses down along the hammer handle (Arteria manubrii mallei). This last named vessel descends from the upper wall of the canal bound up in the cuticular strip referred to previously as the bande cutanée of Til-

laux. During its downward course behind the Manubrium on its way to the umbo it approaches gradually nearer the Manubrium.

The writer takes exception to this generally accepted view as to the situation of the Arteria manubrii mallei. He finds that the vessel does *not* run *behind* the manubrium. It merely appears to do so from the fact that it descends along with the bande cutanée which lies behind the Stria malleolaris, but not behind the hammer handle, for there is quite as much of the hammer handle behind, as in front of the Stria malleolaris. More about which will be said later on, together with its clinical significance. According to Schwalbe

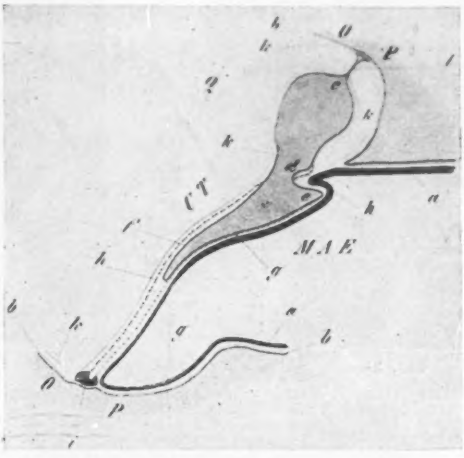

Fig. 20. Taken from Troltsch. Beitrage zur Anatomie des menschlichen Trommelfells, pages 91 to 98. Zeitschrift fur Wissenschaftliche Zoologie. Band 9, 1858; tafel VII, A; fig. 2. Schematic vertical section through the hammer and drum membrane. MAE, external auditory canal; CT, drum cavity; OP, temporal bone; a, epidermis and cutis of the external canal extending onto the drum head; b, periosteum; c-f, hammer; c, head; d, neck; e, short process; f, hammer handle; g, radial fibre layer; h, circular fibres in front and behind the hammer; i, tendinous ring; k, mumous membrane covering of the drum cavity, the circular fibre layer, the hammer and the superior ligament of the hammer.

the artery divides at about the level of the lower third of the manubrium into two branches, an anterior and a posterior. The former courses in front of the manubrium, while the latter runs behind and more downward. According to Moos, both may remain separated or form a loop-like anastomoses about the umbo. No matter whether they remain separated or form the loop, numerous small radiating branches are given off. These again divide up into capillaries. Between the smaller branches and the capillaries are found tiny loop-like anastomoses between neighboring radiating branches

(secondary Bogen of Moos, which were also observed by Prussak in his examination of the drumhead of dogs). A few small radiating side branches form loops which anastomose with a venous plexus. These latter in turn collect into veins of increasing size and finally empty into two large veins which course in an upward direction along the manubrium to eventually reach the upper wall of the canal.

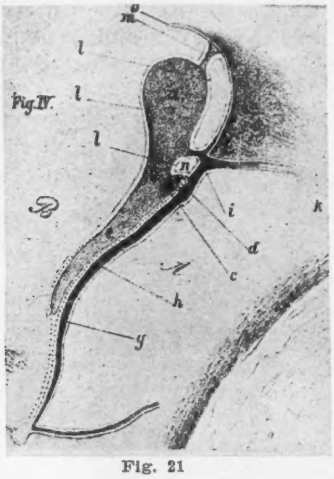

Fig. 21

Fig. 22

Fig. 21. Taken from Prussak. "Zur Anatomie des menschlichen Trommelfells". Archiv. fur Ohrenheilkunde, Band 3, 1867, pages 255 to 279, tafel II, fig. IV. Schematic picture of the arrangement of the layers of the drum membrane about the hammer and a view of the upper pocket of the drum head from the side. A. Meatus auditorius externas; B, drum-cavity; C, tympanic margin of the squamous bone; a, hammer head; b, hammer neck; c, short process of the hammer; d, its cartilaginous part; e, hammer handle; f, circular fibre layer of the Tunica propria; g, radial layer; h, process of the cuticular element of the external auditory canal; i, layer which covers the upper pocket of the drum head from above; k, epidermal layer; l, mucous membrane layer of the drum cavity; m, Lig. suspensorium mallei; n, cavity of the upper pocket of the drum head; o, periosteum.

Fig. 22. Taken from Gustav Brunner, 1870. "Beitrage zur Anatomie und Histologie des mittleren Ohres", tafel 2, fig. 16. Section through the long axis of the hammer, perpendicular to the drum head; A, bony canal, upper wall, Rivine's notch; B, hammer head; C, hammer neck; D, hammer handle; E, short process; F, Membrane flaccida; G, Ligamentum externum mallei; H, Chorda tympani; I, tendons of Tensor tympani; K, Prussak's space; a, cartilage; b, drum membrane fibres; c, dermal layer of the drum head; d, discontinuat; e, Haversian canals; f, smaller marrow spaces.

Most of the radiating arterial side branches course toward the periphery of the drumhead and finally form a capillary network around the margin of the drum membrane. This arterial capillary network anastomoses with a venous network on the cuticular surface of the drumhead at the periphery which is in communication with the veins along the manubrium. Anastomoses occurs also on

Fig. 23

Fig. 24

Fig. 23. Taken from Politzer. Lehrbuch der Ohrenheilkunde, 1908, page 27, fig. 38. Longitudinal cut through the hammer, the drum head and the outer attic of the tympanic cavity; h, hammer head; te, Annulus tendinosus; b, Processus brevis mallei; u, umbo; t, Chorda tympani; l, Ligament mallei sup.; Ligament mallei ext.; s, Membrane flaccida (Shrapnell); ae, attic ext.; P, Prussak's space; c, blood vessels between the external attic and the bony canal.

Fig. 24. (Author's). A photographic reproduction of a more or less frontal section through the temporal bone. The hammer is sectioned longitudinally. The specimen is presented here in order to show the free space between the lateral aspect of the hammer handle and the membrane. This particular section is just posterior to the Stria malleolaris.

Fig. 25. (Author's.) Section is posterior to the previous one. This section shows admirably well the wide space between the hammer handle and the drum membrane.

the external cuticular layer of the drumhead between the arterial vessels which radiate from the center and those which run a converging course toward the umbo. Finally there is a through and through communication between the peripheral arterial network on the external layer and the internal mucous membrane layer previously referred to. According to Moos the larger veins collect blood from the surface of the drumhead and then course upward along the manubrium. According to Prussak the return blood from the cuticular layer of the drumhead follows according to the relatively greater negative pressure in one or the other of two directions (a) toward the hammer handle, or, (b) toward the periphery of the drumhead.

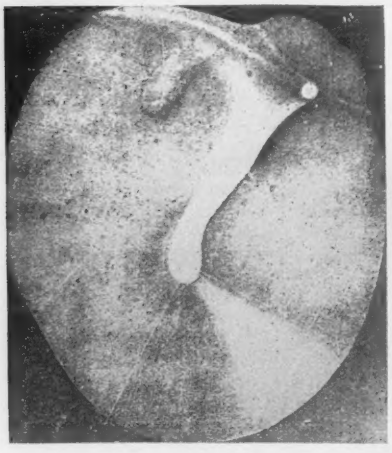

Fig. 26

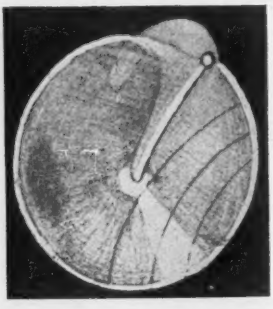

Fig. 27

Fig. 26. Otoscopic picture of the right drum head. From Politzer's Atlas.
Fig. 27. For description see figure 28.

The blood supply of the mucous membrane layer of the drumhead comes from the Annulus tympanicus. It forms on the Stratum mucosum of the drumhead a capillar network which is radially arranged. The arterial blood of this network is supplied principally from a small artery which descends in the mucous membrane of the hammer handle. A branch is given off which goes to the periosteum of the median part of the manubrium. There is, in addition, arterial blood supply which comes up from the floor of the tympanic cavity to supply the lower periphery of the drumhead and anastomoses with the branches of the mucous membrane artery of the hammer handle, according to Moos. The venous return like-

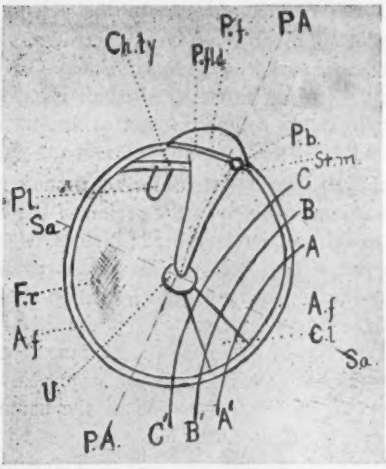

Fig. 28. Original schematic representation of the right drum head. Pf, Pars flaccida; Pb, Processus brevis of the malleus; Pl, Processus longus of the incus; Pfld, posterior fold or plica of the drum head called by Politzer the posterior stripe of Prussak. The anterior fold extends from the Processus brevis to the Spina tympanica anterior of Henle and represented in the sketch by a very short double line not more than one-sixth as long as the posterior. All authorities agree that this anterior fold is shorter than the posterior but no one has ventured a difference to be nearly so great as here represented. For instance, in Politzer's Atlas, figure 26 in this paper, the anterior fold is represented a trifle shorter than the posterior; the same holds true for the illustrations of Bruhle, Parson, Phillips, Alexander, Toldt and Spalteholz. From otoscopic estimates taken of numerous drum heads the writer finds that the distance from the short process of the hammer to the anterior spine is never more than one-third as long as the distance from the short process to the posterior spine, more often it is not more than one-fifth. The dashed line PA running lengthwise, with the hammer handle represents the primary axis which divides the membrane into two unequal halves, an anterior and a posterior. The posterior half is normally a trifle larger than the anterior. The difference is perhaps more marked than is here represented. The dashed line Sa represents the secondary axis passing through the lower tip of the hammer handle and at right angles to the primary axis. The secondary axis divides the drum head into a superior and an inferior half. The superior is slightly larger than the inferior. U, umbo, the small navel-like area of greatest depression where the tip of the hammer handle is firmly fixed to the drum head with an increased amount of interlacing fibres of connective tissue principally radiating. It is because of the greater abundance of stroma fibres at this location that causes the membrane here to appear whiter and more opaque than elsewhere with the possible exception of the Annulus fibrosus. Af, Annulus fibrosus. Extending around the greater part of the periphery from the anterior to the posterior spine; Ch and Ty. Chorda tympani nerve discernible as a cord of varying dimention depending upon the thickness of a remnant of embryonal connective tissue wrapped about the nerve. Ordinarily it has a sufficient tinge of pink to give it a pale fleshy color. The Chorda tympani nerve is discernible in about 8 per cent of individuals who have normally translucent drum heads. St.m, Stria malleolaris, extending from the Processus brevis along the hammer handle to the lower tip. It divides the hammer handle into two unequal halves. It is this line which separates the anterior part of the external surface of the hammer handle, which is in intimate contact with the drum head and the posterior part of the hammer handle which is not in intimate contact. See text. Cl, cone of light, the brilliancy of which aids us in determining whether the drum head is smooth or uneven. In the former case the high light appears brilliant; in the latter case it is dull, in some few cases where the surface is extremely uneven the cone of light is absent altogether. The curved line AA' represents the convexity of the anterior inferior canal wall in the average case; BB' represents the convexity of the anterior inferior canal wall in a case of a more pronounced curvature of the canal than represented by AA'; CC' case of extreme curvature of canal where one finds it quite difficult to examine even the anterior boundary of the hammer handle.

wise follows two directions (1) about the margin of the drumhead, where there is found a venous network, and (2) in the mucous membrane about the hammer handle. Here begins the formation of a vein which carries the venous blood upward, annexing a vein coming from the periosteum of the median surface of the hammer handle.

The veins of the Stratum mucosum anastomose with the veins of the Stratum cutaneum, both in the region of the manubrium and also about the margin of the drumhead; besides, in the intermediate zone venous branches perforate the Membrana propria and permit thereby communication between the circulation on the inner and outer layers of the drumhead according to the description of Moos. The many smaller veins of the mucous membrane about the region of the hammer handle collect to form fewer large veins, which ascend until they reach the level of the neck of the hammer when they perforate Schrapnell's membrane and unite first with veins which come from the Sulcus tympanicus and still further on with the veins of the posterior wall of the canal.

The inclination of the drumhead to the external bony canal is fairly well represented in Figs. 15 and 16, taken fom Schwalbe, Fig. 17, taken from Politzer; Fig. 18, from Toldt;[23] Fig. 19, from Spalteholz.[22] It is generally understood that the inclination of the drumhead varies considerably in different individuals, but so does the contour (curvature) of the canal. The writer finds that the plane of the drum membrane varies less than does the curvature of the canal. In fact, the angle between the two structures is dependent more upon the curvature of the canal than upon the inclination of the drumhead. In other words, the contour of the canal determines more than any other single factor the acuteness of the angle formed by the junction of the anterior inferior wall of the canal and the drum membrane. At the same time it determines for us whether it is difficult or easy to examine otoscopically all that portion of the drumhead which lies anterior to its primary axis. However, we are more accustomed to thinking in terms of inclination of the drumhead than of curvature of the canal. The more pronounced the curvature of the canal, that is to say, the smaller its radius of curvature, the sharper is the angle formed by the lower anterior wall of the canal with the drumhead and the less we are able to see the anterior inferior margin of the drumhead.

In a horizontal section of the temporal bone, Fig. 15 (Schwalbe Fig. 159), one can see the acute angle formed by the drum membrane with the anterior wall of the canal. This angle is estimated

to be from 45° to 55°. In a vertical section, Fig. 16 (Schwalbe, Fig. 160), one can see the angle formed by the drum membrane and the inferior wall of the canal. The angle will be found to be somewhat less than 40°. Bezold[26] estimates the angle formed by the drumhead and the anterior inferior wall of the canal at 37° 35′. This wedge shaped acute angular space in the lumen of the canal has been designated by Bezold as the Recessus meatus auditorii externi, and by H. Meyer[25] as the Sinus meatus.

Fig. 29. Taken from Spalteholz Hand Atlas der Anatomie des Menschen. Band 3, 1903, page 805, fig. 885. Right drum membrane.

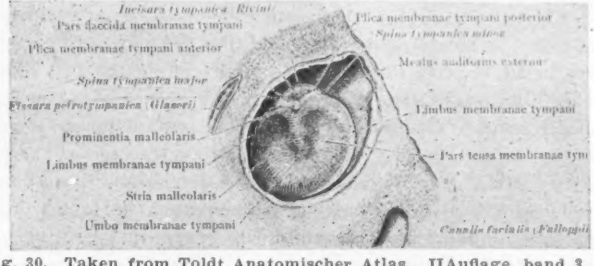

Fig. 30. Taken from Toldt Anatomischer Atlas. IIAuflage, band 3, 1921, page 924, fig. 1420. The external surface of the left drum head.

The drum membrane does not presnt a simple plane surface, but is distinctly funnel shaped. To be sure, the funnel is very shallow, and its concavity is presented externally. Figs. 17, 18, 19; also 20, taken from Tröltsch, and 21 from Prussak. The last two figures are so much alike that it suggests to one the possibility that Prussak's illustration was taken from Tröltsch's. Neither does the margin of the membrane lie in a single plane. On the contrary it is distinctly spiral formed; as a result, the insertion of the membrane posteriorly and superiorly is more laterally situated than the anterior inferior insertion.

The greater part of the descriptive anatomy of the drumhead up to this point has been taken from Schwalbe, who in turn abstracted the most of what he presents from works of the original investigations in the field of anatomy, human and comparative. The present writer adopted the same plane. In other words, he followed up the literature and obtained as far as possible the data herewith presented from first-hand sources, retouching the descriptions as he felt the need. In those few instances where he does not accept the views of others he so puts the case as to leave no doubt as to which are the views of others and which are his own. Not to present the anatomy of the drumhead as set forth by original investigators of marked ability would be to do them, as well as the subject, a grave injustice. Besides, the writer by presenting his own interpretation, after having given that which is generally accepted, allows a better opportunity for contrasting differences wherever they exist.

The essential purpose of the present paper is to bring out the otoscopic appearance of the normal drumhead and its behavior under certain physical tests, leaving to some future occasion a discussion of its appearance and behavior in those various pathologic processes of the drumhead and drum cavity to which these structures are subject. Before one can accomplish this essential purpose an exact knowledge of the anatomy is necessary; however, the anatomy as accepted by others and thus far presented, does not answer in all respects the purpose. Appreciating the fact that no one can interpret accurately the otoscopic appearance and behavior of the drumhead in health without first possessing an accurate knowledge of the anatomy, both gross and microscopic of the part. the writer looked into the anatomy of the drumhead from as many different sources as was available to him; when he found that in some important details the anatomy as generally taught did not balance with the otoscopic appearance. The disparity which he found existing was just what he anticipated. However, it needed confirmation. The greater divergence of opinion as to the anatomy of the drumhead existing between other anatomists and the writer concerns the manner of fixation of the hammer handle to the drum membrane. The popular idea concerning the subject is that which is presented by Schwalbe, whom the writer has liberally quoted. Schwalbe represents the cross-section of the hammer handle throughout the greater part of its length, elliptical in shape, with one sharp edge presented toward the membrane. The writer takes exception to this. Brunner's interpretation comes nearer to that of the writer; however, they are not the same. In Brunner's[20]

illustration, Fig. 12 of this paper, is shown an angular space posterior to the line of fixation of the hammer handle to the membrane filled in with a scroll of connective tissue. This angle he calls the posterior discontinuät. In Fig. 13, cross-section of hammer handle at a lower level than the preceding, he leaves a small free space behind the attachment of the hammer handle in what he terms the posterior discontinuät. His representation of this figure showing the angular space between the posterior half of the hammer handle and the drumhead external to it, is far more accurate than Schwalbe's description, or illustrations. Brunner's monograph was written as early as 1870. This detail which the present writer accepts as important was missed by Schwalbe and practically everyone else since. Fig. 22, in this paper, taken from

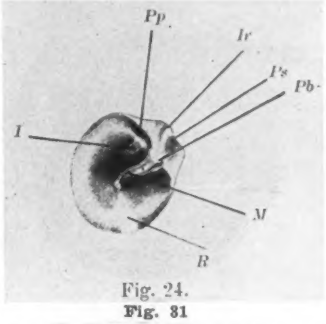

Fig. 24.

Fig. 31 Fig. 32

Fig. 31. Taken from Prof. Dr. G. Alexander. Die Ohrenkrankheiten im Kindesalter, 1912, page 19, fig. 24. Normal drum membrane. M, Malleus; Pb, Processus brevis; Ps, Plica superior; I, Incus; R, Cone of Light; Ir, Incisura Rivini.

Fig. 32. Taken from Wendell C. Phillips. Diseases of the Ear, Nose and Throat, 1922, page 176, fig; 101. The normal Membrana tympani.

Brunner's Fig. 16, represents a longitudinal section through the hammer handle and the drum membrane. This section furthermore shows a space between the hammer handle and the membrane. The fact that he has the space represented dark in color would suggest that he believed the space to be occupied with connective tissue agreeing with his cross-section of the hammer handle and drumhead. Politzer in his text-book, Fig. 38 (see Fig. 23, this paper) also presents an illustration of a longitudinal section through the hammer handle and drumhead. He shows a distinct space as Brunner does, but contrary to Brunner he has left the space open. Politzer makes no especial mention of this space in spite of the fact that it is represented in the illustration. The writer presents two halftone microphotographs from his own collection which distinctly

show a free space between the hammer handle and the drumhead. These sections are both longitudinal ones through the hammer handle posterior to the Stria malleolaris. Figs. 24 and 25.

Referring to this space from another angle the writer recalls distinctly that Professor G. Alexander as long ago as 1907 was wont to point out the fact that in the presence of acute exudative catarrh the hammer handle appears to be narrower than normal. He did not go so far as to venture an explanation for it. The writer has been able on repeated occasions to confirm this observation of Alexander and has come to adopt this finding as pathognomonic of exudative catarrh. He did not venture an explanation for this finding until some time after he began using a magnifying electric otoscope, when by the use of the Siegle attachment, he observed

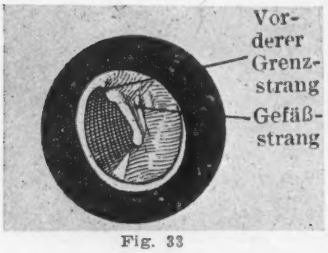

Fig. 33

Fig. 34

Fig. 33. Taken from A. Passow. Trommelfellbilder, 1912, tafel IV, fig. II. Normal left membrane.

Fig. 34. Taken from G. Bruhl Ohrenheilkunde, band XXIV, page 99, fig. 48. 1, short process; 2, hammer handle; 3, umbo; 4, light reflex; 5, anvil-stirup articulation.

with the aspiration of air from the external auditory canal, that is, by producing negative pressure, that that portion of the drumhead which lies directly over the posterior part of the hammer handle, in other words, that portion which lies behind the Stria malleolaris, was lifted away from the hammer handle. Later on when examining cases of so-called chronic middle ear catarrh with relaxed drumheads, it was possible to observe with the same magnifying Siegle otoscope a lifting of the drumhead directly behind the Stria malleolaris from the underlying posterior half of the hammer handle far enough to cause the appearance of overlapping, every time aspiration was used. Besides, in those cases of pathologically increased fixity of the ossicles (including the hammer), the drum membrane was observed to move by aspiration and compression of air with the Siegle otoscope more freely than the hammer handle,

giving rise to a picture quite similar to that observed in cases of pathologically relaxed drumheads. In some cases of pathologically relaxed drum heads the outward excursion of the drumhead produced by aspiration was so exaggerated as to permit the membrane to overlap the anterior half of the hammer handle as far even as its anterior margin. The appearance of relaxation was exaggerated somewhat owing to the obliquity of the posterior superior half of the drumhead, which allows one a better opportunity to ob-

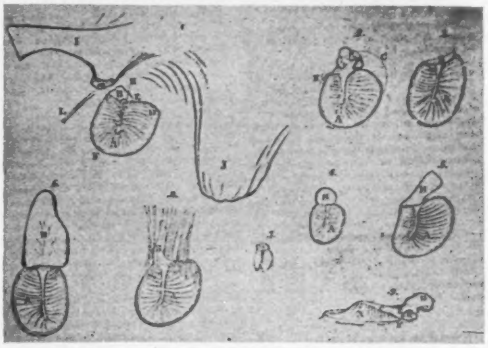

Fig. 35. Taken from Shrapnell on "the form and structure of the membrana tympani", pages 120 to 124. London Medical Gazette, vol. X, 1832, page 124, fig. 1. External view of the membrana tympani, left ear. A, Membrana tensa; B, Membrana flaccida; C, anterior superior angle; D, posterior angle; E, process between the angles; F, oval circumference with a second line introduced to show the extent of the groove; G, external surface of the malleus, to which the Membrana tensa is not attached; H, tubercle at the base of the manubrium of the malleus; I, zygomatic process of the temporal bone; J, mastoid process; K, portion of the base of the zygomatic process sawn off; L, situation of the glenoid fissure.

Fig. 2. Internal view of the membrana tympani, right ear. A, membrana tensa; B, Membrana flaccida much developed; C, dotted line completing an oval figure; E, Processus gracilis entering the glenoid fissure.

Fig. 3. Cat; fig. 4, rabbit; fig. 5, dog; fig. 6, sheep; fig. 7, rat; fig. 8, ox; A, Membrana tensa; B, Membrana flaccida; same in fig. 3 to 8 inclusive. C, in fig. 8, appearance of muscular fibres in the Meatus externus; fig. 9, profile of the internal surface of the Membrana tympani; A, Membrana tensa; B, Membrana flaccida; C, manubrium; D, head; E, neck; F, tubercle of the Os malleus.

serve the phenomena because of the parallax. The opposite holds true for the anterior inferior half of the drumhead, since here we are viewing the drumhead more perpendicularly to the surface.

Besides the posterior, Brunner refers to an anterior discontinuät. From the manner in which he puts it in the text and illustrates it in the several cross-sections of the hammer handle, he believes there is an angular space between the anterior margin of the hammer handle, which he represents as free in the tympanic cavity, and the drum head. He refers to this as the anterior discontinuät. The

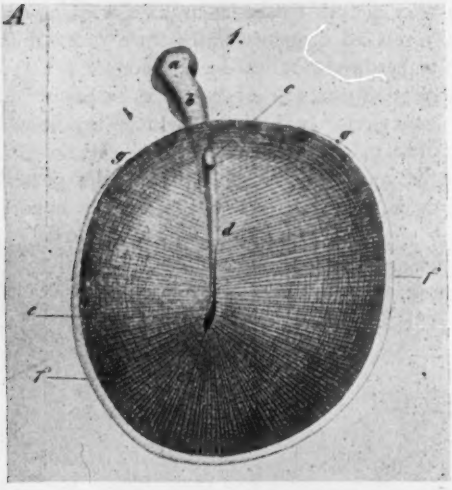

Fig. 36. Taken from Troltsch. "Beitrage zur Anatomie des menschlichen Trommelfells", pages 91 to 98. Zeitschrift fur wissenschaftliche Zoologie, band 9, 1858, tafel VII. A, fig. 1, schematic view of the drum head from the external canal after removal of the external layer; a-e, hammer; a, head; b, neck; c, short process; d, hammer handle whose anterior edge serves for the attachment of the radial connective tissue fibres; e, spatulated end of the hammer handle; f, tendinous ring (Annulus cartilaginous).

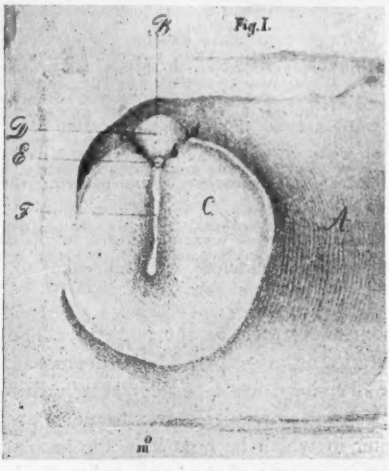

Fig. 37. Taken from Prussak. Zur Anatomie des menschlichen Trommelfells". Archiv. fur Ohrenheilkunde, band 3, 1867, pages 255 to 279. tafel II, fig. I. The outer surface of the drum head of the left ear. A, posterior wall of the external canal; B, tympanic margin of the squamous bone; C, drum head; D, Membrana flaccidi Shrapnelli; E, Processus brevis; F, hammer handle; a, anterior and b, posterior fold which separates the drum head proper from the Membrana flaccida; c, the anterior and, d, the posterior angle between the Membrana tensa and the Membrana flaccida.

writer does not believe that such a space exists; instead, he believes that the anterior margin of the hammer handle is in intimate contact with the Stratum fibrosum (middle layer) of the drum head. Furthermore, he believes that this intimate union of hammer handle and drum membrane reaches from the anterior margin of the hammer handle to a line somewhat less than half-way posteriorly, and from the short process of the hammer to the spatulated extremity of the hammer handle. The writer is convinced that that part of the drum membrane which lies posteriorly to the posterior line of attachment and external to the lateral aspect of the hammer handle is not in contact with the hammer handle. Furthermore, the farther posteriorly we follow the external surface of the hammer handle behind its attachment to the drumhead, the deeper into the tympanic cavity it reaches. In other words, the anteriorly directed wedge shaped space between the drum membrane and the free posterior half of the lateral aspect of the hammer handle behind the posterior attachment of the hammer handle first described by Brunner, is *not* filled with connective tissue as he suggests, but open and free. The result is that the posterior half of the hammer handle is less well defined by otoscopic examination than the anterior half. Its diminished visibility is comparable with that produced by looking through ground glass at an object a short distance beyond the inner surface. The farther an object is from the ground glass the less distinct in outline it appears when viewed through the opposite side and incidentally the larger it appears.

In Fig. 26, representing the otoscopic appearance of the drumhead, taken from Politzer's Wand Tafeln, the writer on a former occasion ran a faintly white chalk line along the posterior margin of the hammer handle to partly obscure it and make it appear more like what as the writer believes it naturally does. He left it thus modified in the illustration herewith presented.

The writer presents also an original more or less schematic drawing of th right drumhead, Fig. 27. The posterior margin of the hammer handle is represented faintly as it ought to be. The hammer handle at the spatulated end is firmly fixed to the drum membrane with an extra supply of interlacing fibres, which because of their greater abundance and their peculiar arrangement tend to produce a white, more opaque halo about the spatulated extremity of the hammer handle.

Superiorly and posteriorly the osseous wall of the external auditory canal continues in a more or less curved direction medianward and downward onto the drum membrane and continues on the membrane in the same general direction as far as the umbo region.

This continuously curved line formed by the superior wall of the canal and hammer handle down to the umbo is well shown in the illustration taken from Politzer's atlas, Fig. 17. It is well to bear these anatomical relationships in mind, for in the case of the mildest periostitis of the external superior canal wall, secondary to a mastoid empyema, the coincident swelling of the periosteum has the effect of producing in place of the normally continuously curved line or surface an angular depression at the Annulus. The position of the Annulus has not changed, but the swelling of the drum membrane anteriorly and inferiorly and of the canal wall posteriorly and superiorly produces the effect of a furrow between them at the Annulus tympanicus. This sign when present even to the slightest degree is pathognomonic of mastoid involvement, according to the writer's experience.

Besides the knowledge one is able to obtain of the anatomy of the drumhead from the gross and microscopic study of the same, something can be added by way of careful otoscopic studies, especially with the aid of the Siegle attachment and magnifying lens, and, we might add too, a study of the drumhead before, during and after inflation by the Politzer or catheterization method, according to the needs in the case.

The writer has been in the habit of listing the findings of the normal tympanic membrane as follows:

1. The membrane should be *intact.*
2. It should be *brilliant.*
3. It should be *normally translucent.*
4. It should be neither retracted nor bulging, in which case we find the *superior half larger than the inferior* and the *posterior half larger than the anterior.*
5. It should be *normally mobile with the Siegle instrument.*
6. It should be *normally mobile with Politzer inflation.*

Let us next consider each of these qualities in the order in which they have been presented.

Naturally we would expect to find the normal drumhead intact. A question arises—is it possible to find the tympanic membrane not intact, yet appearing as though it were? The answer is yes. It is quite possible for a perforation in the anterior inferior quadrant to exist without being visible. It can occur in those cases where there is an excessively curved canal, where the anterior inferior wall of the canal is elevated excessively. In other words, where there is a very acute angle formed by the junction of the anterior inferior wall of the canal and the anterior inferior part of the drum head. Failure to detect a perforation in this location is worth

mentioning for the reason that it happens every now and then, more especially in the experience of the tyro, when Politzer inflation will aid one in detecting a dry, whistling noise in the presence of a dry perforation, or the bubbling sound in the presence of secretion, providing the Eustachian tube is patulous. Where secretion is present the inflation will cause the secretion to well up into the canal, often mixed with air bubbles.

Brilliancy: The normal drumhead is brilliant because it presents externally a smooth polished surface. The brilliancy is most easily recognized by the character of the cone of light. So long as there is no acute congestion or inflammation involving the drumhead or drum cavity the drumhead will appear smooth and, therefore, brilliant. A few of the less expert otologists seem to confuse dullness with opacity. The drumhead in a so-called chronic middle ear catarrh, in the absence of an acute exacerbation, is brilliant. In cases of this kind at times the opacity is mistaken for dullness. Again, it is possible to find a healed chronic middle ear suppuration presenting a brilliant membrane. The writer interprets a brilliantly appearing drum membrane as evidence of the absence of any acute pathologic process of the drum membrane or drum cavity and conversely the appearance of *dullness* as positive evidence of *acute* involvement of one or the other of these structures. Dullness of the drum head as a physical finding is comparable to dullness of the cornea. This comparison is very interesting, but belongs to a separate chapter and for another occasion. All other things being equal, dullness of the membrane due to the physical unevenness of the surface tends also to make a normally translucent membrane more opaque just as effectually as does dullness of the cornea from glaucoma tends to decrease the transparency of the cornea. Carrying the comparison a little farther, just as increased intra-ocular tension tends toward the production of edema of the corneal epithelium (steamy cornea), so does increased intra-tympanic pressure produce a dullness of the drumhead.

The normal translucency of the drumhead is rather difficult to define. The normal drum membrane is not transparent like glass, neither is it opaque like paper. It is less transparent than the former and decidedly more transparent than the latter. We can at best describe it as translucent, tending decidedly more toward transparency than opacity. It is the presence of the connective tissue fibres of the middle layer that prevents the membrane from being more transparent than it is. On the other hand, the arrangement of the fibres, that is in more or less parallel running layers, (comparable with the arrangement of the connective tissue fibres

of the corneal stroma) make for greater transparency than would otherwise occur. Normally, the drumhead is sufficiently translucent to permit one to see indistinctly the posterior margin of the hammer handle and the long process of the incus, the latter a trifle less distinctly than the former. In exceptional cases one can make out, indistinctly, the nearer crus of the stapes. In other cases one can see indistinctly the niche of the round window as a dark shadow through the posterior part of the drumhead.

Since there is a greater amount of connective tissue fibres about the Annulus fibrosus and about the umbo region we find these two locations more opaque and whiter than the remainder of the Pars tensa. For this reason the writer has been making use of these more densely appearing white areas as contrasting guides from which to judge the relative opacity of the remainder of the drumhead. For instance, if the contrast is marked the membrane on the whole must be less opaque than when the contrast is only slight. Summing it up, if one can see (1) the posterior edge of the hammer handle and (2) the long process of the anvil indistinctly and (3) the Annulus fibrosus and the connective tissue about the spatulated end of the hammer handle in marked contrast with the rest of the drumhead the membrane may be considered normally translucent.

On the other hand when the Pars tensa becomes opaque from the presence of organized exudate on its median surface as happens after repeated attacks of acute exudative middle ear catarrh, then the opacity of the drumhead prevents one from seeing the posterior half of the hammer handle and the long process of the anvil. The hammer handle presents an appearance of narrowing and the posterior line of attachment of the hammer handle shows up more sharply than under normal conditions.*

Opacity of the drumhead from the presence of organized exudate on its median surface is not the only condition that can produce an obscuration of the posterior half of the hammer handle and thereby an appearance of narrowing. The same general appearance of the hammer handle may result from the presence of a fluid exudate in the middle ear cavity. In all such cases the opaque fluid fills up the wedge shaped space in the tympanic cavity between the posterior lateral aspect of the hammer handle and the drum membrane.

*It must be borne in mind that the posterior line of attachment of the hammer handle to the drum head is not the same as the posterior margin of the hammer handle. The former corresponds fairly well with what some of the writers refer to as the stria malleolaris which runs somewhat anteriorly to the midway line between the anterior and posterior edge of the hammer handle.

As to the color of the drumhead, it is much more difficult to describe than its translucency. The writer has never found a painting of the drumhead sufficiently accurate to be acceptable; whereas in his own atempts he has failed even more signally than others. The color is influenced by two factors (1) the color of the normal mucous membrane of the inner wall of the tympanic cavity and (2) the normal translucency of the membrane through which one views the inner wall of the tympanic cavity. Although the color is difficult to reproduce in a painting it is not at all difficult to recognize after having been trained to seeing it. For one who is familiar with the normal coloration of the drumhead, or rather of the median wall of the tympanic cavity, viewed through the normally translucent drumhead, it becomes a comparatively easy matter to recognize the very pale yellow or straw color of muco-serous exudate behind the drumhead or to recognize that trace of pinkness to the promontory, which is characteristic of otosclerosis.

The normal drum membrane should be neither retracted nor bulging. The absence of either permits the posterior half to be slightly larger than the anterior and the superior slightly larger than the inferior.* In the event of retraction even of slight degree there occurs a reversal of the relative size of these different halves. For instance, the posterior half becomes smaller than the anterior and the superior smaller than the inferior.

In the case of bulging of the drumhead; for instance, from a severe acute otitis media before spontaneous rupture, the direction of the hammer handle remains in about its normal position and measured from this there is practically no change in the relative size of the halves nor is there any exaggeration of the normal difference. The posterior half of the drumhead may bulge, but it does not cause the hammer handle to be displaced any more anteriorly than is found in the normal case. The posterior superior quadrant may appear larger than normal and in a way it is, but only bcause it is appreciably stretched. In fact this stretching of the drumhead with coincidental thinning may cause the posterior superior quadrant to overlap the anterior margin of the hammer handle. That the bulging, stretched drumhead should overlap the anterior edge of the hammer handle is clearest evidence that the *hammer handle itself does not change its position materially from that found under normal conditions.*

Normal mobility of the drumhead with the Siegle instrument: To understand thoroughly what this really is and what it means one must study the behavior of the drumhead with this instrument

*By the upper half of the drum head in this case is meant the upper half of the pars tensa.

for himself, just as one must do with any other problem which he may want to master. The matter of relative mobility of the drumhead is one of clinical importance. This fact becomes more apparent to anyone the farther he goes into it and especially when he comes to compare the mobility of the drumhead after this method (alternate aspiration and compression of air in the external auditory canal) with that after the inflation method. The normal drumhead is elastic enough to permit one, when aspirating with the Siegle otoscope to discern a movement of the drumhead externalward a slight but definite extent. One can see normally the drumhead (especially the posterior half for reasons given elsewhere) sucked outward with the aspiration when a high light appears, crescentic in shape, near the upper posterior margin. Besides, there is a distinct appearance as though that portion of the drumhead which covers the posterior half of the lateral aspect of the hammer handle is lifted away from the hammer handle more than it is under the normal condition of rest; however, there is no lifting of the membrane forward of the line of the Stria malleolaris as occurs in the case of pathologically relaxed drumheads. On the other hand, by compression one can see that the drum membrane moves inward and with its inward movement there is a disappearance of the high light which was visible near the posterior superior margin of the drumhead, observed during aspiration. The extent of outward motion by aspiration and the inward motion by compression is very slight, but definite. Perhaps all told the complete excursion is appreciably less than 1 m.m. (roughly estimated); at the same time one can observe by parallaxing, taking a fixed point along the external auditory canal, that the hammer handle moves with the membrane. This last mentioned fact is important, for in pathological conditions where there is an undue amount of fixation of the ossicles the excursion of the hammer handle by aspiration and compression with the Siegle apparatus may be observed to be more limited.

In those cases of so-called chronic middle ear catarrh with very relaxed drumheads the excursion of the drum membrane is often observed to be decidedly in excess of the normal, when there occurs more or less overlapping of the relaxed drumhead over and in front of the anterior part of the hammer handle, even beyond the anterior margin of the handle.

The study of the mobility of the drumhead while inflating the middle ear cavity after the Politzer method is quite as important as the study of the mobility with the Siegle instrument. Taken

together, these two methods are of decidedly more value to the otologist than the study of the mobility after either method alone.

A study of the mobility during attempts at inflation of the middle ear cavity tells us something of the relative patulousness of the Eustachian tubes. Generally speaking the greater the outward excursion of the drumhead by inflation of the middle ear the more patulous the tube seems to be. There is a qualifying factor, however, that must not be lost sight of and that is the amount of air pressure applied to the nasopharynx during the attempt at inflation. For instance, if the inflation is a mild one we would not expect (all other things being equal) the same degree of outward movement of the drumhead as when inflation is stronger. There remains still another factor and that is the timing of the inflation; for instance, a strong air pressure administered at the wrong moment is less effectual in inflating the middle ear than a milder one administered at the proper moment. It is because of these modifying factors, which to the writer appears to be very important, that he has come to adopt the plan of having someone experienced to administer the inflation while he studies the behavior of the drumhead during the inflation. The assistant recognizes the amount of resistance felt by the hand during the compression of the bag and announces no resistance, slight resistance, or pronounced resistance, as the case may be. If the assistant who is using the bag calls off to the observer at the scope that there was fair resistance and the observer notes but questionable or very slight outward excursion of the drumhead as compared with the pronounced outward movement, noted earlier by aspiration with the Siegle instrument, it does not require any stretch of the imagination in arriving at the conclusion that the Eustachian tube in this particular case must be abnormally narrow, especially when repeated observations, using the same methods, show consistently these same differences in mobility behavior. If this same case, following dilatation treatment of the Eustachian tube, shows improved hearing and with it an increase in the outward excursion of the drumhead by Politzer inflation we are warranted in concluding that the Eustachian tube is more patulous than it was before the beginning of treatment. The writer will avoid any further discussion of pathologic cases at this time.

Normally the drum membrane moves outward during inflation of the middle ear and about the same degree by this positive pressure exerted in the middle ear after the Politzer method as by negative pressure applied to the external canal with the Siegle instrument.

The normal elasticity of the drumhead permits it to go back to its

primary position after inflation almost instantly or within a few seconds at most. If it is delayed, or if it requires several acts of swallowing to bring it back, it may be considered pathologically relaxed or else the tube is pathologically narrowed. A confirmation of suspected pathological relaxation is manifested whenever by the strongest inflation there is the slightest degree of overlapping of the drumhead beyond its normal attachment to the hammer handle.

As previously stated, a study of the mobility and elasticity of the drumhead and of the relative patulousness of the Eustachian tube, will give the best results when we examine the behavior of the drumhead after the two methods of Siegle and Politzer, rather than after one alone; precisely as we are able to obtain the fullest information about anything the more angles we are able to study it from.

Before closing, the writer wishes to submit, for the purpose of comparison, illustrations of the drumhead as interpreted by other authors than those already included. Figs. 29, 30, 31, 32, 33, 34. Because of their pioneer work in the field of otology and the value of their contributions to the subject of the anatomical structure and appearance of the drumhead the writer presents in closing, illustrations from Shrapnell, Fig. 35, Tröltsch, Fig. 36, and Prussak, Fig. 37.

The original purpose of the writer was to include in this present effort a discussion of the appearance and behavior of the drumhead in so-called middle ear catarrh, however, he finds the paper already too long and will, therefore, postpone considering the subject of the appearance and behavior of the drumhead in chronic middle ear catarrh for some future occasion.

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A CONSIDERATION OF OTITIC BRAIN ABSCESS, WITH SPECIAL REFERENCE TO DIAGNOSIS AND LOCALIZATION. PRESENTATION OF CASES AND SPECIMENS.*

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Many of us have had the serious malady, brain abscess, under consideration and investigation for the past twenty years or more, and in spite of our study of the subject the mortality remains at about 40 per cent, or higher, and were it not for the fact that a fair proportion of our cases are discovered accidentally or by sheer luck, the mortality would be much greater. Today, as formerly, two chief difficulties stand as barriers to a realization of our coveted goal; the first is the serious state of patients applying for treatment, and the second is our faulty and inaccurate methods of localization. The former, the advanced illness of the patient when first seen, continues to be the chief obstacle to a lessened mortality, because the early symptoms and indications are not recognized as primary to an intracranial lesion. Considerable advance has been achieved in the second difficulty—localization—with indications of a progressive betterment.

The limits of this paper are necessarily so restricted that I will confine my consideration of the subject to calling attention to a few important phases, with a view of eliciting an exhaustive discussion. For the same reason I shall confine my remarks to otitic temporosphenoidal and cerebellar abscess.

The vital point that we are called upon to determine is the presence of a cerebral suppuration in its early stages, even though it may be too early to make serious effort at localization. This is quite possible, even in slowly developing cases and before the evidence of compression becomes manifest.

In the *initial stage* we have headache, fever, chills, or chilly sensations, and periodic vomiting. The symptoms may rapidly subside, which supports an incorrect diagnosis of acute indigestion or abdominal grippe.

The *latent stage* may continue indefinitely, but the patient does not enjoy good health. He has periodic headache, avoids exertion,

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shows changes in physical and mental habits, and becomes increasingly morose and eccentric, until finally, complicating some supposed general illness, he suddenly shows marked symptoms of an intracranial lesion, and is ushered into the *stage of manifest symptoms*, chiefly general debility, weariness, loss of appetite and flesh, constipation, and a general feeling of malaise, and sometimes convulsions in children.

Headache is one of the most common and important symptoms, this notwithstanding the fact that the patient may have been subject to head pains. The pain may range from a dull headache to the most acute suffering and may involve any part of the head, from the temporal region to the occiput, depending on the location of the abscess, or it may be general and have no reference to localization. If localized, it is not necessarily confined to the side of the aural infection. In only one or two instances have I been able to elicit pain on percussion, as has been suggested by some authors. In one case the headache had continued intermittently for a period of several months before the chilly sensations developed, the mother believing that this headache was peculiar to the child and had nothing to do with her progressive illness, as she (the mother) had always suffered from what was termed "a woman's headache". At operation this case proved to be an encapsulated abscess.

Although subnormal temperature is distinctly characteristic of a brain abscess formation, it must be remembered that this occurs only when the process does not involve the dura. It is not uncommon to have more or less of a rise in temperature when the suppurative process works its way toward the surface and produces a localized involvement of the meninges. Later, as the process becomes walled off, the temperature will again become about normal. I have seen several of these cases where the erosive process has penetrated the dura, discoloring the tegmen tympani, and in other instances the bone itself has been absorbed, providing for ample drainage, as shown by a subsidence of practically all symptoms. In two cases we would not have suspected an intracranial abscess had it not been for the previous history. So long as the circulation is not invaded by pathogenic micro-organisms, the temperature will remain about normal or even subnormal. A persistent subnormal temperature, pulse and respiration are indicative of a deep latent abscess which has not involved the adjacent brain structure.

In the presence of an otorrhea, especially of the chronic type, the chilly sensation frequently spoken of with little thought of its

significance, is often the beginning of an intradural suppuration. In one of my cases these chilly sensations continued for a period of weeks, but it was noted by the patient's mother that their beginning also marked a definite change in the child's health, which grew progressively worse. On the assumption that malaria was responsible, the patient received no actual medical care until eventually a pronounced chill, lasting twelve to fifteen minutes, together with other suggestive symptoms, brought the patient to the hospital.

If the otorrhea of an otitis media becomes scanty or suddenly ceases, and at the same time head pain and nausea or vomiting are present, we should suspect intradural pus formation. This presumption is accentuated if a morose patient suddenly becomes talkative or vice versa.

During the development of an abscess the vomiting is intermittent, not severe, is usually looked upon as due to an error in diet, and does not become projectile in type until pressure symptoms make their appearance. Projectile vomiting without relation to the taking of food is more common in cerebellar than in temporal abscess, accompanied by subnormal pulse, temperature and respiration.

Convulsions, especially when associated with other indicative symptoms, are almost pathognomonic of intracranial abscess formation, especially in children suffering from a chronic otorrhea. In my experience, convulsions are common in children, but very infrequent in adults. Disturbances of the sensory and mental state are always present. At first the patient is apathetic and drowsy, yawns, answers questions slowly and with hesitancy. Periods of alertness and clearness of mind alternating with an apparently unconscious state, are followed by a failing memory, then mental dullness, ending in stupor and coma; the former may continue for some time and this is the opportune moment for operation, before the later—and terminal—stage intervenes. The child whose brain is here for your inspection suddenly fell to the ground in a severe convulsion, while playing out of doors. Although she had had chilly sensations and suffered from headache for some weeks, this severe seizure was the real incentive that led to a recognition of the intracranial disease from which she sought relief. The abscess was situated in the temporosphenoidal lobe. A detailed report of this case follows:

History: R. W., female child, age 7 years. First attack of earache four and a half years ago, with spontaneous rupture of mem-

brana tympani and prompt recovery. Three years later, complicating influenza and pneumonia, she suffered from multiple abscesses of the arms, back, right hip, and left occipital region, which continued to drain for five months. Nine months later, Dec., 1922, there began severe head pain, increasing in intensity until August, or eight months, when it was relieved by spontaneous rupture of right membrana tympani, the same ear in which spontaneous rupture took place four years previous. Associated with this were facial palsy and bulging of right eye. For three weeks prior to entering hospital, in Sept., 1923, there was frequent projectile vomiting, and on entrance the patient was in a semi-conscious state and greatly emaciated.

Operation: Mastoid operation immediately performed. Tegmen antri showed no evidence of disease. On account of symptoms, a large surgical exposure of dura was made. Dura firm, not discolored. Transmitted pulsation as though the impulse passed through a firm mass. Incision through dura made and a large temporal-lobe abscess, containing more than two ounces of pus, evacuated.

Post-operative Progress: Patient returned to full consciousness after coming out of ether and was able to retain food. She gradually gained weight and improved in general health. Her condition was so generally satisfactory that I exhibited her to my student class, feeling that she had recovered from her brain abscess. Several weeks later there was a gradual return of lack of interest in her surroundings, and finally a sudden attack of vomiting, a convulsion, high fever and almost immediate death.

Until the acute exacerbation this patient ran a temperature ranging from normal to slightly subnormal.

The neurological examination showed palsy of the third right nerve, with slightly exaggerated reflexes on the left side.

The eye-ground showed swelling and opacity of disc before operation, lessening considerably after operation, but vessels remained quite tortuous. Left eye practically normal.

X-rays were negative.

Blood cultures, both day before death and at autopsy, were negative.

Smear pus removed at time of operation showed many pus cells and gram positive cocci.

Cultures of pus showed staphylococcus albus and bacillus pyocyaneous.

Autopsy: Entire temporal lobe of brain found to be necrotic. In its central portion was a well defined abscess cavity 5x3x3 cm. in size.

The following brief report illustrates the expression often heard that a person suffering from a chronic otorrhea is practically living over a volcano:

C. P., female, about 45 years old, had had discharge from each ear practically all her life, but enjoyed excellent health. Seven months ago, after finishing her domestic duties, she went out to do the family marketing, but before reaching her destination fell unconscious to the pavement, was picked up by a policeman and taken to Memorial Hospital.

A cursory examination showed a well-nourished, apparently healthy woman, with temperature of 98°. Eyes reacted normally to light; pupils were equal and of normal size. Each membrana tympani was destroyed and the canals filled with pus.

Examination of urine and blood showed a considerable amount of sugar.

Although unconscious, the patient resisted movements of the arms but not of the legs. There was intermittent vomiting. Reflexes were normal.

Six hours later the pupils were dilated and did not react to light. Leucocytosis, 30,000. Temperature 102.3°. Differential not noted.

On account of the presence of sugar it was thought that the patient was in a state of diabetic coma, in the absence of all evidence of injury. She was, therefore, referred to Dr. Orlando H. Petty, who, during his examination, noted the discharging ears and suspected at once a brain abscess. On her way to the operating room the patient died without regaining consciousness.

Autopsy findings: Extensive organizing clot covering left temporal lobe and lower central part of left hemisphere. Localized abscess about the size of a quarter in the anterior portion of the left temporal lobe. No visible destruction of the floor of the left middle fossa about the middle ear. Diagnosis of cerebral hemorrhage left temporal region following an abscess of the left temporal region. Complication of left suppurative otitis media.

In two other cases of temporosphenoidal abscess in children, a long-continued headache and finally convulsions were the only symptoms noted up to the time of operation.

One of the characteristic symptoms of a developing abscess is the progressive ill health shown by a coated tongue and loss of appetite and weight. The latter is especially marked when the cerebellum is

involved. Given, therefore, a case of suppurative otitis media presenting chilly sensations, headache, vomiting, changed disposition, general malaise and loss of flesh, the indications are that the patient is suffering from an intradural abscess formation. Indeed, only a few of these symptoms combined are sufficient to make us suspect the presence or beginning development of such a process and should warrant a thorough investigation.

When the symptoms are obscure, or even temporarily absent, the facial picture of one suffering from an intracranial abscess gives the impression of marked distress from serious illness.

Intracranial pressure is present to some extent in brain abscess formations and is quite sufficient ultimately to cause a reduction in the temperature, pulse and respiration. This should not be confused with the extreme pressure produced by tumors. The latter cause pressure by an actual increase of new tissue formation, whereas the former cause pressure at the expense of broken-down brain tissue, which necessarily must be relatively moderate in degree.

While choked disc is a characteristic symptom of intracranial pressure from any cause, and therefore not indicative of the causative factor, its presence should be determined and considered in connection with other leads.

Optic neuritis, when present, is diagnostically helpful and may be bilateral or confined to the affected side; its absence has no significance.

I wish again to record my experience that abscess formations complicating chronic otorrhea happen much more frequently in the recurrent type. In these cases the abscess forms slowly, is usually walled off, protecting the surrounding brain structure, and may exist for a long time without attaining sufficient size to cause compression symptoms.

Generally speaking an increased leucocytosis is a reliable index of the body resistance, while an increase in the polymorphonuclear percentage indicates the severity of the infection. However, conclusions obtained from blood counts should not be hastily accepted. Some patients become gravely ill without material increase in either, while others with a high leucocytosis and polymorphonuclear percentage are not always seriously sick.

The chief point of value is the maintenance of a relative ratio between the two, so that when the polymorphonuclear percentage is high the infection is counteracted by a corresponding increase in the leucocytosis. An increasing polymorphonuclear percentage with a falling leucocytosis is a bad omen, whereas the reverse is an indi-

cation of a favorable outcome. The leucocyte count and the polymorphonuclear percentage are usually higher in meningitis than in brain abscess or sinus thrombosis.

All varieties of organisms in pure culture or mixed infection may be present in otitic brain abscess; those most commonly found in the acute and subacute types are the staphylococcus aureus and the streptococcus pyogenes. The colon bacillus has been found in a majority of my chronic cases.

When localizing signs reasonably determine the presence of an abscess, the area should be explored even though the dura is normal in appearance and the brain pulsates, as would be the case when the lesion is deeply situated, and it follows, per contra, that it is unwise to assume the non-existence of an abscess in the absence of the dull gray dura and non-pulsating brain which are present when the lesion is near the surface.

The same principle holds true in the presence of a collection of pus between the bone and dura. If the evacuation of this extradural collection does not promptly relieve the symptoms, intradural exploration is imperative. It is quite legitimate to suspect an accumulation of pus within the cranial cavity in the presence of a profuse otorrhea not reasonably attributable to the existing tympanic and mastoid disease, even in the absence of general or localizing symptoms. I have seen several of these cases where the pus escaped through a carious opening in the tegmen tympani and was not suspected before operation.

An otitic intracranial abscess is usually situated close to the surface of the brain, in immediate propinquity to the area of diseased bone through which the infection has spread. When more deeply located the infection is not transmitted by contiguity but through the blood vessels or lymphatics.

Should intracranial symptoms disappear following the mastoid operation and then recur, an exploratory operation is immediately indicated.

Wherever the otitic intracranial lesion may be located ultimately, I have frequently noted enlargement of the deep cervical glands as the first observable symptom. This primary glandular enlargement is usually overlooked but is sufficiently constant as an indication of intracranial disturbance, in the presence of an otorrhea, to warrant careful consideration.

Although rigidity of the neck is one of the prominent symptoms associated with meningitis, it is also common to a certain extent in brain abscess cases, since a majority of them also suffer from a circumscribed meningeal involvement.

Involvement of the fifth nerve is shown by lessening of the corneal reflex and facial hyperesthesia, and that of the twelfth nerve by deflection of the tongue.

Involvement of the pyramidal tract is indicated by disturbances of the special senses of smell and taste, and a hemiplegia involving the face, arm and leg with increasing severity in the order mentioned.

It is well to keep in mind that otitic abscess formation is much more frequently found in the temporosphenoidal lobe on the side of the coexisting aural disease, so we are justified in operating at this point in case of doubtful localizing symptoms, and, furthermore, however confirmatory or even characteristic the symptoms may be, the fact remains that sinus thrombosis, subdural abscess, meningitis, and even pyemia are much more likely to be present than brain abscess, and that meningitis is the most frequent intracranial complication of an acute otitis.

In order to give the patient the best possible chance for recovery it is essential that we should determine the exact location of the abscess formation at the earliest possible stage. The primary focus of infection is, therefore, very important in our attempts at localization. According to published records temporosphenoidal abscess predominates over cerebellar abscess in children in the proportion of about eight to one, and approximately two to one in adults. As the large majority of otitic abscess formations are found in the temporosphenoidal lobe, we should first endeavor to eliminate the cerebellum. The cerebellum can be reasonably excluded if we are able to produce vertigo and past-pointing by stimulation—turning or caloric, preferably the former. The cerebellum may become involved either through the labyrinth or by way of the sigmoid sinus. It is reasonable to assume that if the labyrinth is involved the abscess formation is in the cerebellum. This is emphasized if the patient presents a staggering gait and projectile vomiting.

If our tests show the labyrinth to be intact the otogenous abscess is likely to be found in the temporosphenoidal lobe. These general principles are valuable and yet are not definitely conclusive and should be considered in their bearing on other manifestations.

Indications of a temporosphenoidal abscess are symptoms of compression, subnormal pulse, temperature and respiration, paralysis of the opposite side, drowsiness, aphasia when situated on the left side. In some cases, when pressure is exerted on the auditory center, there is deafness on the opposite side.

It is easy to visualize the pathology of a temporal otitic abscess when we remember the histologic changes through which a chronic

mastoiditis passes. Eventually the granulation tissue and other inflammatory debris filling the mastoid cavity are converted into hard, ivory-like bone which effectually resists the erosive action of destructive pathogenic organisms, and excludes all external or cortical evidence of the underlying disease. Quite naturally the necrotic process now follows the line of least resistance and invades the interior of the skull via the thin tegmen antri or tegmen tympani, which fall an easy prey to the ravages of the invading micro-organisms when denuded of their mucosal lining.

Due to the fact that the temporosphenoidal lobe is the silent area of the brain, I have seen large collections of pus accumulate in this locality virtually without symptoms. Aphasia, more particularly that type which may be designated as object-naming aphasia, is one of our important diagnostic symptoms. One of my cases had object-naming aphasia when he entered the hospital. Indeed, it was owing to the fact that his parents thought that he became mentally deranged at times that the attending physician had him admitted to the hospital on the chance that his otorrhea might be a causative factor. This patient knew definitely every object that was held before him, but was unable to name any of them. He recognized a key and pointed to the door, turning his hand as though unlocking it; he indicated a knife by going through the motions of whittling; a nail-file, by indicating its use. A large temporosphenoidal abscess was evacuated, with practically immediate relief of his word-naming aphasia.

Word-aphasia in a right-handed patient suffering from a chronic otorrhea of the left side is pathognomonic of abscess involving the left temporosphenoidal lobe.

High temperature, pulse and respiration are characteristic of the acute invasive stage. In one of my recent cases, these symptoms, together with severe headache, led the attending physician to suspect a meningitis, in the presence of a discharging ear. In the course of two or three days all symptoms except the head pain subsided, leading to the belief that the illness was due to gastro-intestinal disturbance. Very shortly, however, symptoms of compression, including aphasia, led to a realization that an intracranial lesion existed, which proved to be a temporal-lobe abscess.

In the absence of localizing symptoms, slow pulse and respiration and low temperature, together with a persistent headache, would lead us to suspect involvement of the right temporosphenoidal lobe.

Not any of my cases have presented "word deafness", as so admirably described by Eagleton in his recent work on "Brain Ab-

scess" (p. 178). This is true also of hemianopsia, which in all probability was overlooked on account of its transitory nature and the infrequent ophthalmological examinations, as pointed out by Eagleton.

A facial palsy which develops rather slowly, in the presence of an otorrhea, and involves the opposite side of the face, is quite characteristic of temporosphenoidal abscess, especially when accompanied by headache and repeated chilly sensations. When the extremities are involved, this also is of a progressive nature.

Recurrent attacks of stupor offer a valuable diagnostic point in the early stages of temporosphenoidal abscess. These become more pronounced and finally continuous as the expanding lesion of that lobe progresses.

The development of hallucinations of various types in the presence of an intradural lesion, of which much has been written, indicates involvement of the temporosphenoidal lobe. As compression increases the mentality markedly changes, slowness of speech and comprehension are replaced by heedlessness or mental aberration. Periods of somnolence may suddenly change to a state of mental excitation simulating delirium.

Cerebellar abscess is much more difficult to diagnose, especially in the early stages, than is an abscess of the temporal lobe. I remember seeing, many years ago, the post-mortem findings of a case of destruction of the cerebellar hemisphere which came under the care of a general surgeon, in which practically no localizing symptoms were present. As before stated, early manifestations are frequently observable in temporosphenoidal abscess, but symptoms that are more or less characteristic of cerebellar abscess commonly do not occur until pressure symptoms show themselves, such as vertigo, paralysis of the face and extremities of the same side, projectile vomiting, staggering gait, spontaneous pointing deviation, spontaneous vertical nystagmus, falling, and lateral deviation of the eyes.

Barany has pointed out that if there is rotary nystagmus toward the affected side and no longer any irritability of the labyrinth, one can be reasonably sure of the existence of a cerebellar abscess, provided the patient has a slow pulse and low temperature. As the disease progresses nystagmus gradually increases and is always more marked toward the affected side.

If symptoms of intradural suppuration are present and tests reveal a dead labyrinth or the presence of a lateral sinus thrombosis, the indications are in favor of the abscess being located in

the cerebellum. The patient is inclined to fall towards the opposite side from the lesion, and this, with optic neuritis, will serve to differentiate this condition from internal ear suppuration, in which the gait is of a swaying or staggering character, without optic neuritis.

We must recognize that the stimuli for muscular co-ordination and tonicity originate in and are controlled by the functional activity or inactivity of the cerebellum. Therefore, any deviation from the normal, such as muscular paralysis (which, however, would not be complete unless the pyramidal tract was involved), would indicate cerebellar involvement.

In a male patient of about 35 years, I noted a rapid loss of flesh, together with an absence of the patellar reflexes and very frequent projectile vomiting, all of which disappeared in due course on evacuation of the cerebellar abscess.

Satellite abscess formations are not infrequent. Those which have come under my observation at post-mortems did not communicate with the parent abscess and in all probability were the cause of the death, as under some circumstances a fatal issue is not dependent on the bulk of the abscess.

However important it is to operate at as early a stage as possible, we must keep in mind that during the progress of advancing softening the formation of pus will not yet have occurred and it will be most unwise to attempt evacuation at this time. The difficult point is a proper recognition of just when an abscess formation is complete, and at the same time this clear conception of its development is most important, as above stated.

Great stress is often placed upon the presence of optic neuritis, which may be found in a few days after the onset of the illness; in other cases it does not appear until the disease is well advanced. If we are reasonably convinced of the presence of a lesion we should not wait for the appearance of optic neuritis.

The very best time for surgical intervention is during the early purulent stage, when the high fever and severe general headache have largely subsided and the stages of stupor and irritability have been replaced by the sleepy state, where cerebration is slow, the intellect dull, and temperature and pulse are just about normal, with pupillary inequality. Any delay after this favorable time for operation lessens the patient's chances of recovery in increasing degree. To wait for the arrival of the toxemic or paralytic period, when pupils may be either dilated or contracted and deep coma ensues, offers the patient poor chances of recovery, even in

the hands of the most skillful operator. Unfortunately, this is the stage in which many cases are received, and this adds largely to our mortality percentage.

We must always be cautious in our statements as to the permanent cure of a patient suffering from an abscess in any part of the brain. Even so, one of my patients, a child of nine years, in whose case I evacuated pus from the temporosphenoidal, then the cerebellar, and finally from the frontal lobe, was operated upon in 1909, more than fifteen years ago. This case was reported in THE LARYNGOSCOPE, Aug., 1910. She is now in normal condition, mentally and physically, and is married and the mother of two healthy children. She is here for your inspection this evening.

Another patient, a male, 37 years of age, was also operated upon in March, 1909, and a temporosphenoidal abscess evacuated. He enjoys excellent physical and mental vigor today and is present for examination.

A third case is that of an adult 42 years old, from whose brain I evacuated a temporosphenoidal abscess twelve years ago. He is a hard-working man and enjoys good health. He is present tonight.

A fourth brain abscess case was operated upon seven years ago. She made a good recovery, has continued well and is present tonight. Her history has not been published and is given below:

M. W., female, aged 30 years. Admitted to hospital, June, 1917. Five weeks before admission patient had pain in right ear for several days, followed by spontaneous rupture of the membrana tympani. At the end of this period the discharge suddenly ceased, with almost immediate swelling over the mastoid process. The usual routine examinations were negative, with the exception of the ear.

At operation a carious opening was found over the cortex. The cells were completely destroyed and the cavity filled with pus and some granulation tissue. Medium-sized perisinus abscess. Nothing in the tegmen antri nor tegmen tympani to indicate any intracranial lesion. Post-operative temperature for six days, 99° to 100.2°; then normal. Patient left the hospital twelve days after operation, apparently in good condition.

Three weeks after mastoid operation readmitted to hospital, suffering from headaches, and vomiting after meals. Attacks of vertigo when walking, with tendency to fall towards the affected side. An examination of ears was negative, but there was some swelling of the deep cervical glands on the right side, and a moderate but progressive loss of flesh. Patient was in a semi-comatose state,

talked irrationally, with convulsive movements of the extremities and a widely dilated right pupil, the left being normal. Some redness over the scar of previous mastoid operation. Otherwise examination negative.

At operation the tegmen tympani was discolored and when removed revealed a bulging and tense dura. On incision about four ounces of greenish-yellow pus was liberated, the abscess cavity being from 2½ to 3 inches deep. At the time of patient's second admission temperature was at all times either normal or subnormal, and so continued throughout the progress of her recovery.

Some time after the second operation this patient suffered from convulsive movements, which were pronounced not to be Jacksonian in type. She has now completely recovered. I regret that the laboratory reports in this case have been mislaid and are not available for record.

This patient is present for your inspection.

I believe we can reasonably say that these four cases have permanently recovered. On the other hand, some of my brain abscess cases have died suddenly, some time after operation, in what were termed convulsions, presumably from a refilling of the pus sac, or an existing satellite abscess which may have ruptured into the ventricles.

I am indebted to Dr. Lewis Fisher for the detailed report of the three cases following, in which it was his good fortune to obtain Barany tests prior to operation. Not any one of these cases presented symptoms of an intracranial lesion other than the Barany reactions.

Case 1: C. F. Chief complaint, discharge right ear. Dizziness, first attack two years ago, last attack two weeks before examination. Patient able to work. No other symptoms.

Examination showed a chronic purulent otitis media, right side, with large central perforation. Total deafness. Hearing was good in left ear, and appearance normal.

Vestibular examination revealed no spontaneous phenomena deviating from the normal. Turning tests showed nystagmus after turning to the right and left of 24 and 12 seconds, respectively, suggesting a non-functioning right horizontal canal. The vertigo was diminished but past-pointing especially poor, the left arm persistently pointing in the wrong direction. The caloric test showed that the entire right ear was practically non-functioning. The left, when douched, showed no response whatsoever for the vertical semi-circular canals, but a good active response for the horizontal.

Patient was advised to enter the hospital but refused, insisting he was not sick and would like to continue with his work.

He appeared for another examination a month later and findings were substantiated at same, with the exception of vertigo after turning, which was considerably diminished, and the right ear, previously non-functioning, did show some trace of responses this time. Patient was again advised to enter the hospital and again refused.

The complete neurologic examination carried out the same day was entirely negative.

Three weeks later patient was brought into hospital in a semi-conscious condition and died the same night.

Autopsy: Autopsy revealed an abscess in the cerebellum on the right side. This patient showed no clinical signs indicating such a grave lesion. The vestibular examination was the only means of suspecting the true condition. The absence of responses from the vertical semi-circular canals but good response from the horizontal canal on the left side (the good ear) definitely indicated a central lesion. The interference with vertigo and past-pointing further suggested that involvement was probably cerebellar. The abscess was deep in the substance of the cerebellum and could not have been found at any ear operation, had one been undertaken, simply on account of the suppurating ear.

Case 2: W. M., male, 40 years old. Examination Apr. 13, 1923. Patient complained of headache for last seven or eight years. Recently slight vertigo. Suppuration in left ear since childhood. Examination showed practically normal-looking right ear, with good hearing. Left ear showed a chronic purulent otitic process, with drumhead gone, the pus fetid and profuse. Hearing quite good. No spontaneous phenomena referable to the vestibular mechanism. Station good. Pelvic girdle movements good; pointing tests performed accurately. Turning to right and left showed good nystagmus, vertigo and past-pointing. Douching the right ear showed a delayed response from the right vertical semi-circular canals. The nystagmus came on very suddenly, in an explosive manner. From the horizontal canal large, active nystagmus.

Douching left, or suppurating ear showed prompt and active response from the vertical semi-circular canals but markedly perverted nystagmus from the horizontal; that is, on tipping the head back the nystagmus, instead of being in a horizontal plane of the head, was vertical and markedly oblique.

We considered the findings indicative of intracranial involvement because the left horizontal canal produced such a markedly perverted nystagmus and the vertical canals of the right, or good ear, showed a much delayed response but one that was marked when it finally did appear. There was hardly anything else to point to a central lesion. There was no choked disc, no palsy of any of the cranial nerves, no weakness of any of the muscles, no sensory disturbances, no ataxia or adiadochokinesia, no fever, no mental symptoms. We considered the vestibular phenomena referred to as sufficiently definite to make a diagnosis of central involvement. The suppurating ear naturally pointed to a brain abscess as being the most likely lesion. Good vertigo and pastpointing after turning excluded the cerebellum, leaving the temporal lobe as the other most likely site for such an abscess.

Operation several days later showed the presence of a large abscess in the temporal lobe.

Case 3: F. L. Reported by Lewis Fisher in "Equilibrium and Vertigo".

Patient had been suffering from headaches and came to the hospital for examination to ascertain whether a previous ear operation, right side, was in any way responsible. Two weeks previously was taken suddenly ill and had no recollection of what happened except that he recovered consciousness in the hospital. Improved in a few days and was discharged practically well except for occasional headaches.

Examination revealed that some mastoid surgery had been done on the right side. Hearing in that ear fair. Left ear entirely normal. No spontaneous phenomena deviating from the normal. On douching the vertical semi-circular canals, both sides failed to respond, while the horizontal canals responded well. This strongly suggested an intracranial lesion. There was very good past-pointing of both arms to the left, after turning to the left, suggesting that the trouble was probably not in the cerebellum. Patient advised to go to hospital but refused.

A week or two later patient again became unconscious suddenly and was rushed to the hospital. The operated ear was again operated upon, without discovery of anything definite intracranially. Neurologic examination entirely negative.

Patient died shortly after, and autopsy showed a large abscess in the temporal lobe, which ruptured into the ventricles, filling the entire ventricular space with pus.

In this case no diagnosis of intracranial lesion could be made on the general clinical phenomena, or, what is more important, even a thorough operative exploration failed to reveal the true condition. The vestibular examination alone indicated intracranial mischief.

Summary: The use of the vestibular tests in the diagnosis of brain abscess may be summed up as follows: They are useful in that frequently they are the only tests that indicate with any degree of definiteness the presence of intracranial involvement. There is nothing about them that would directly suggest an abscess, but what they do indicate is the presence of increased intracranial pressure.

Given a case of unoperated aural suppuration or one where the operation has been performed and the patient is not doing just right, the findings of a spontaneous vertical nystagmus or of a loss of responses from the vertical semi-circular canals, without such a loss from the horizontal canal, or of a perverted nystagmus after douching, etc., at once leads us to suspect very strongly the presence of some intracranial lesion. When the general clinical picture and laboratory tests exclude everything except brain abscess, the vestibular examination might indicate whether such an abscess is in the cerebellum or temporal lobe. The ability to elicit vertigo and past-pointing after stimulation will eliminate the cerebellum and suggest the temporosphenoidal lobe as the probable site of the trouble.

Inasmuch as intradural suppuration does occur without indicative manifestations, it is a wise precaution to determine the labyrinthine reactions prior to all mastoid operations for the relief of a chronic otorrhea, as sometimes, as illustrated by the above cases, the Barany tests give the only clue to an intracranial lesion.

A NEW TECHNIQUE IN THE ROENTGENOGRAPHY OF THE NASAL ACCESSORY SINUSES.

DR. MAXWELL MALTZ. New York City.

I am submitting a preliminary report on a new method of taking Roentgenological pictures of the nasal accessory sinuses. These procedures were developed at Beth David Hospital in the Roentgenological Department of Dr. Charles Gottlieb.

In this method the cassette with double screens is elevated at an angle of 23 degrees. The patient's head is placed in such position that the nose, both lips of the opened mouth, and the chin touch the plate in the center. An ordinary sinus cone is used and the anode of the tube is placed directly over the central part of the vermilion border of the upper lip. A fairly hard tube is employed (gas tube), 35 milliamperes, 4 gap, and an exposure of 3 seconds. The cone is placed snugly against the head. We have been able to obtain slightly clearer pictures by using the Bucky diaphragm at an angle of 23 degrees and giving a 6 second exposure for posterior-anterior views.

In Dr. Waters' position the chin rests on the plate lying flat with the table, the long axis of the tube is parallel to the plate, and the nose of the patient is from 1-1.5 c.m. from plate. The anode of the tube is parallel to the nose tip.

The author's position has the advantage over Dr. Waters' in that:

1. By having the mouth opened, the antrum is stabilized and placed nearer to the plate. In this manner the variability factor of Dr. Waters' chin position is eliminated.

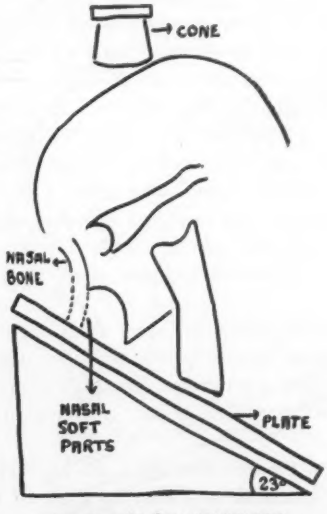
2. In Dr. Waters' position the petrous portion of the temporal bone is seen behind the lower angle of the antrum. The author's position automatically shifts the petrous portion of the temporal bone *below* and behind the antrum. In that way the petrous portion of the temporal bone never clouds the lower part of the antrum.

3. In Dr. Waters' position the outer part of the body of the sphenoid is superimposed upon the lower and inner part of the antrum. In my position there is an advantage in having the mouth opened, in that the sphenoid can be visualized below and behind the thin horizontal portion of the palate bone.

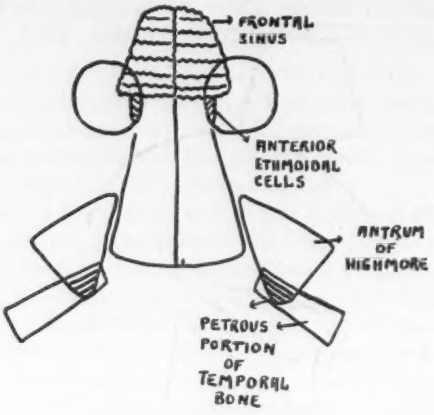
4. In the author's position the X-rays do not penetrate so much thickness of bone as in the Waters' position. Therefore theoretically and practically one obtains a clearer picture in my position. In Waters' position most of the central rays penetrate the front, comparatively thick part of the greater wing of the sphenoid, bone

Editor's Note: This mss. received in The Laryngoscope office and accepted for publication April 8, 1924.

DR WATERS' POSITION



AUTHOR'S POSITION NO I



Dr. WATERS' POSITION
(DIAGRAMMATIC)

near the pterygoid tubercle, then the roof of the spheno-maxillary fissure striking bone in this location at an angle. The rays then penetrate the lower border of the spheno-maxillary fissure striking bone in this vicinity at the apex of an angle. The rays then strike the floor of the antrum.

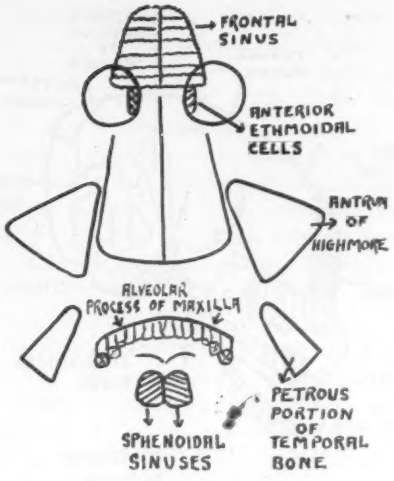
In my position the greater part of the central rays penetrate comparatively thin bone at the back part of the horizontal portion of the frontal. The rays then pass through the floor of the orbit and then strike the floor of the antrum. Here the rays penetrate successive layers of bone that are nearly horizontal.

5. A minor advantage is that in my position, the nose, open mouth, and chin, resting against the plate, offers the patient less opportunity of moving the head and gives one a slightly better chance to obtain both antrums on the same plane.

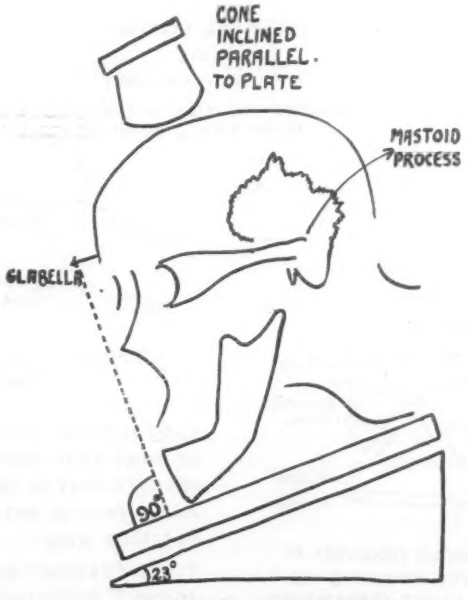
6. In the author's position one can see more of the deeper part of the antrum.

In using an ordinary disarticulating skull for experimental purposes, we have been able to prove the advantage as outlined in point No. 4. In this experiment the rays penetrate bone alone, and in my position the antrums appear clearer than those of Waters' position.

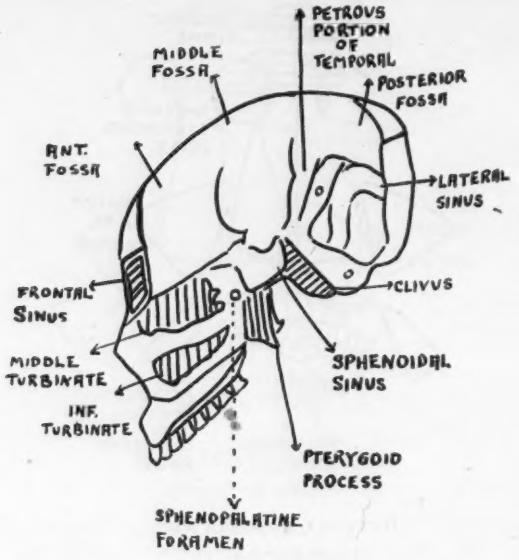
As an auxliary to the new method, I use another method to show concomitantly the sphenoids and another angle of the antrums. In this second method an ordinary 23 degree angle, wooden block is



AUTHOR'S POSITION NO I
(DIAGRAMMATIC)

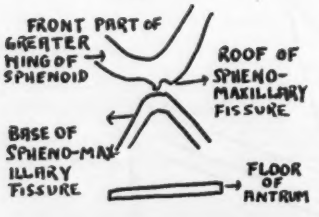


AUTHOR'S POSITION NO II



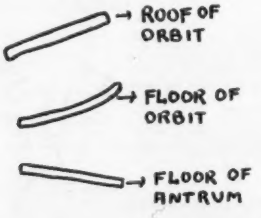
SAGITTAL SECTION
OF SKULL
WITH ROOF CUT AWAY
SHOWING POSITION OF SPHENOIDAL SINUS
IN AUTHOR'S POSITION NO II.

I



COMPARATIVE THICKNESS OF BONE THRU WHICH THE GREATER PART OF THE CENTRAL X-RAYS PASS IN DR. WATERS' POSITION

II



COMPARATIVE THICKNESS OF BONE THRU WHICH THE GREATER PART OF THE CENTRAL X-RAYS PASS IN AUTHOR'S POSITION NO I.

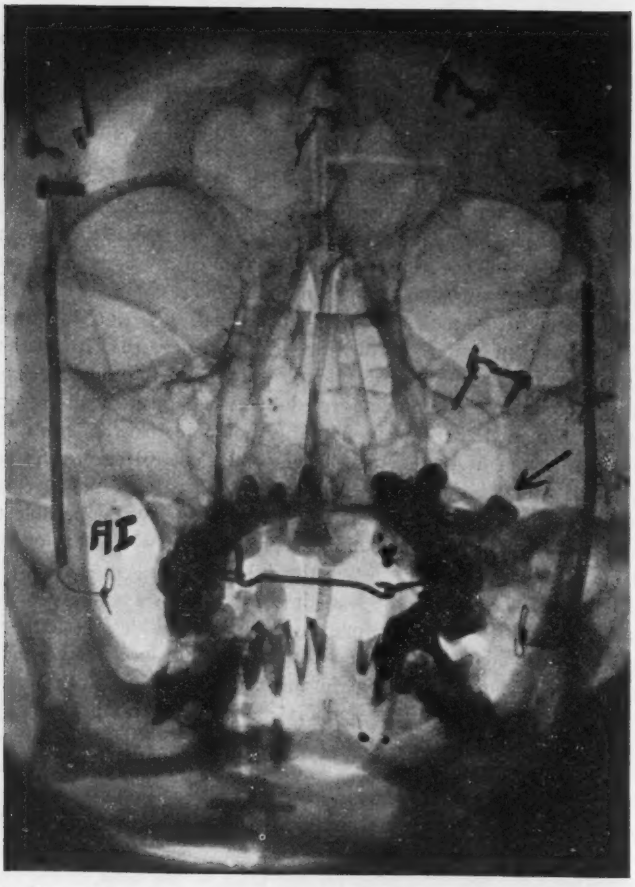
THE DIFFERENT BONES LIE ALMOST PARALLEL TO ONE ANOTHER.

employed. The chin is placed on the hypothenuse and the neck extends over the altitude of the triangle. The chin is placed in such position that the imaginary line drawn from the glabella to the hypothenuse forms an angle of 90 degrees with the hypothenuse. In this wise, the base of the sphenoid lies parallel to the inclined plate. At the same time, due to the declivity of the hypothenuse and the declivity of the base of the sphenoid, mucoid material or pus in the sphenoid will descend to the front part of the sphenoid, where the base is comparatively thinner than the base of the sphenoid posteriorly. In this way one can more readily visualize pathology in the sphenoids.

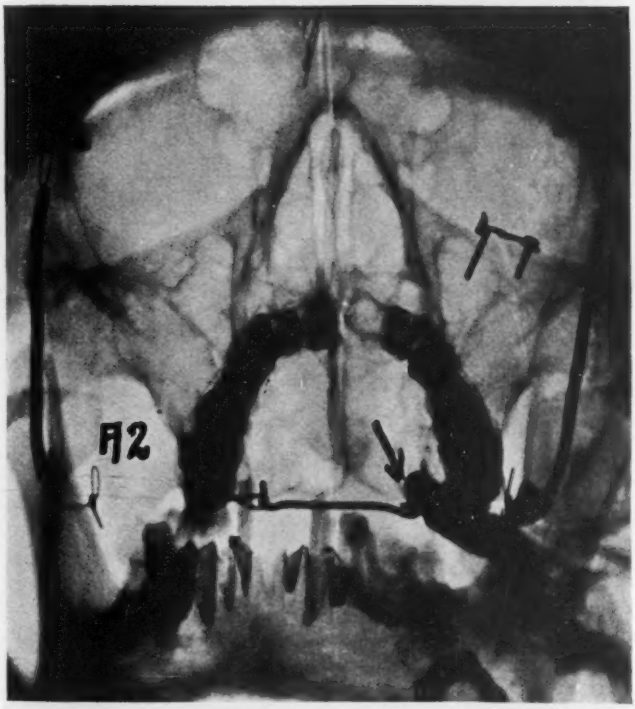
In this second position the sinus cone lies parallel to the hypothenuse, that is, the tube is tilted to 23 degrees. In this case, more of the *external* part of the antrum can be visualized since the base of the antrum is hidden from view by the comparatively broader and more developed alveolar portion of the superior maxillary bone in the region of the molar teeth. Thus, a small collection of mucoid or mucopurulent material which is visualized near the base of the antrum in the first position would, in the second position, mechanically descend into the angle formed by two opposite walls of the antrum, provided there were no large polypoid growths posterior to the collection of pus. And the mucopurulent material, thus descending, would be obscured by the posterior part of the alveolar process of the maxillary bone. In this wise one would be able to study and visualize the mucous membrane upon which the mucopurulent material was resting in the first position.

In the second position the best time exposure is from *3½-4 seconds*. Also, in this position, one can visualize, besides the antrums and the sphenoidal sinuses, the base of the skull and its foramina, such as the foramen spinosum, foramen ovale and the carotid canal in the petrous portion of the temporal bone. One can also visualize the mastoid cells on either side. Thus, this position is of value not only in giving you another view of the antrums and sphenoidal sinuses, but is also of value in clearing up questionable cases of fracture of the base of the skull. Besides, this position can be used as an aid in taking mastoid plates, since here one readily visualizes both mastoid processes on one plate at the same time. This eliminates the variability factor when taking alternately pictures of the right and left mastoid processes. Thus it is advisable to take three pictures for mastoids. Apropos to mastoid technique we obtain the most satisfactory pictures by tilting the *Bucky diaphragm* to an angle of 23 degrees and using an exposure of 4 seconds. The cone lies directly over the temporo-pareital region, and is not filled.

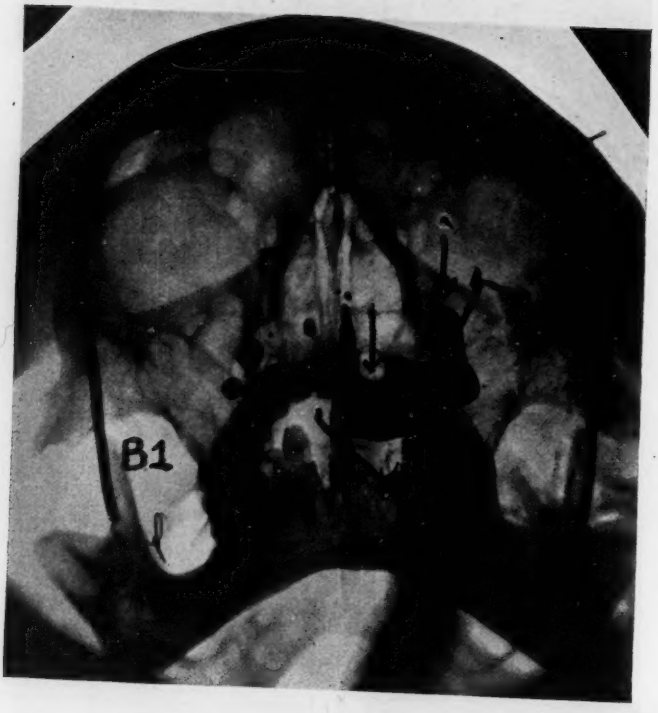
1824 Lexington Avenue.



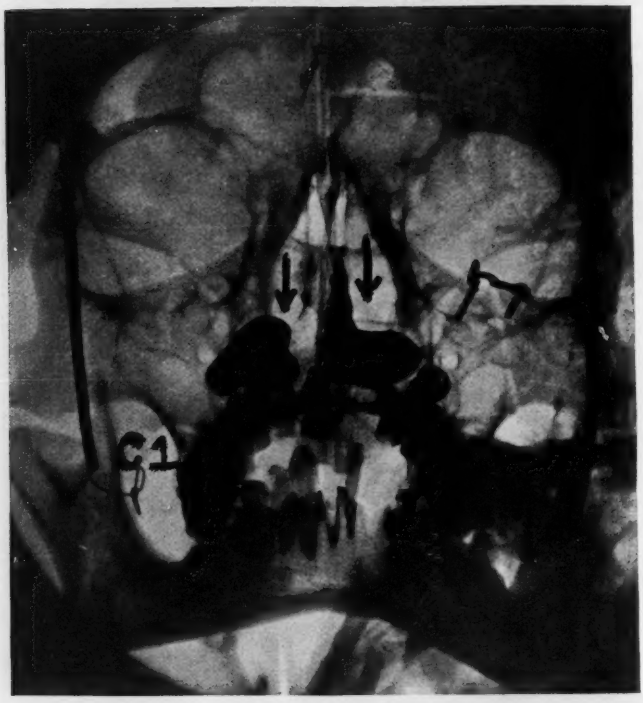
A—D are a series of experimental plates on a disarticulating skull.



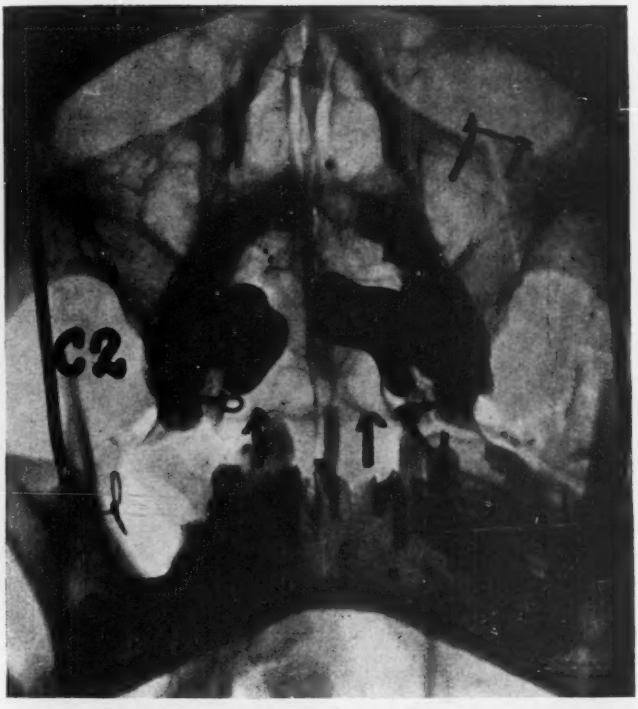
A1 is Waters' position. A2 is the author's position, No. 1. Mercury was injected into the carotid canal of the petrous portion of the temporal bone. In A1 the petrous portion of the temporal bone is seen to lie behind the lower part of the antrum. In A2 the petrosa lies below and behind the antrum and cannot cloud the antrum.


B1

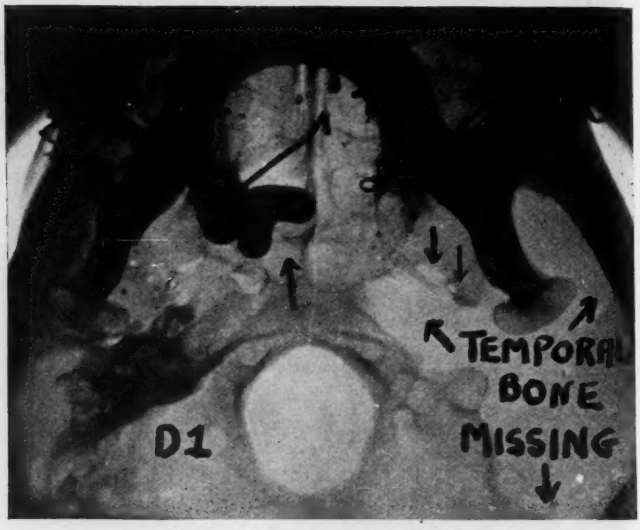
B1 shows mercury in the antrum and in the sphenoidal sinus in Dr. Water's position. The sphenoidal sinus is seen to lie behind the alveolar process of the superior maxilla, and the inferior turbinates. Thus, in the living, the sphenoidal sinuses can never be visualized in this position. B2 is the same experiment in the author's position No. 1. Here the sphenoidal sinus is seen behind and below the horizontal portion of the palate bone. This shows that the sphenoidal sinuses can be visualized in the open-mouth position.



C1 shows both sphenoidal sinuses filled with mercury in Water's position. C2 is the same experiment in the author's position No. 1. This also proves that the sphenoidal sinuses can be visualized in the author's position, and never in Water's position.

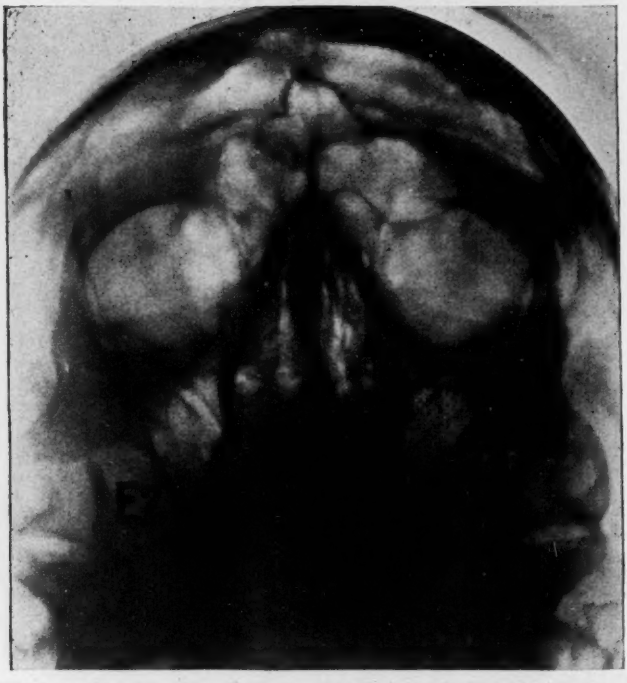


In the A, B, and C plates one can also readily see that the antrums in my position are clearer than those of Water's position, proving that the X-rays penetrate less thickness of bone in the author's position and hence tend to give clearer pictures.

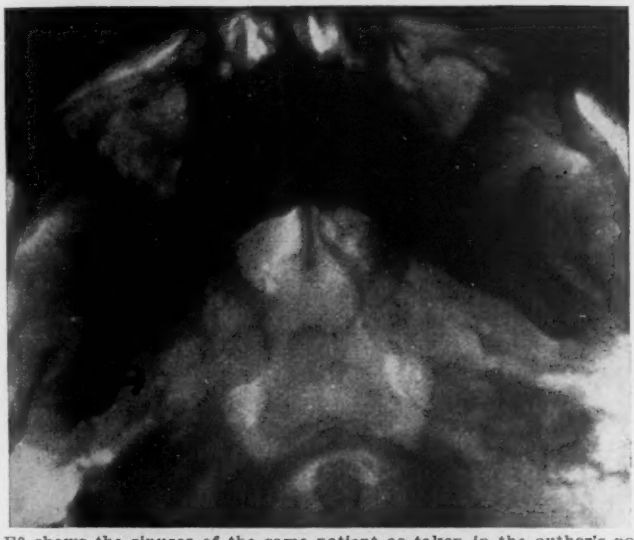


D1 shows one sphenoidal sinus injected with mercury in the author's position No. 2. One also sees both antrums. Some of the foramina at the base of the skull are readily seen. The temporal bone is not present on one side.

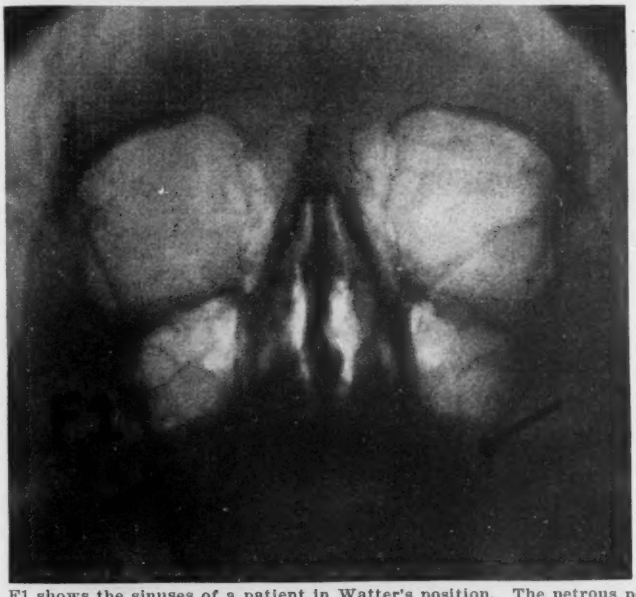
E1 shows the sinuses of a patient in the author's position No. 1. The sphenoidal sinuses are clearly visible. The left antrum is somewhat cloudy.



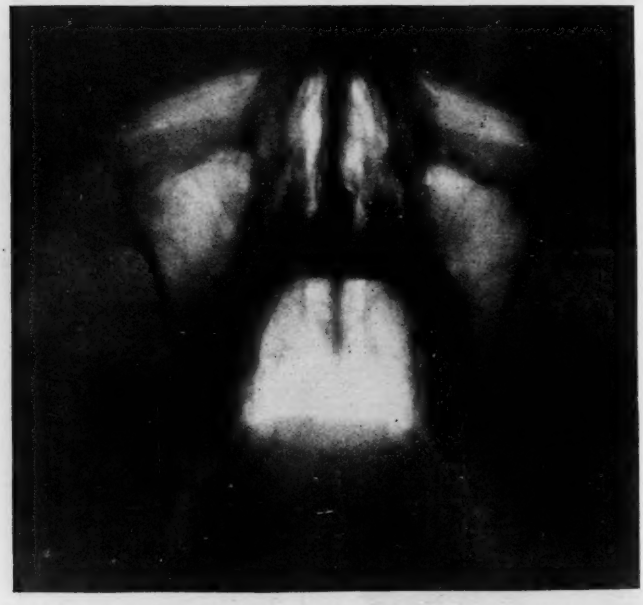
E2 shows the sinuses of the same patient in Water's position. The antrums and frontal sinuses are less clear here than those in the author's position. The left antrum is cloudy. No sphenoidal sinuses can be visible in Water's position.



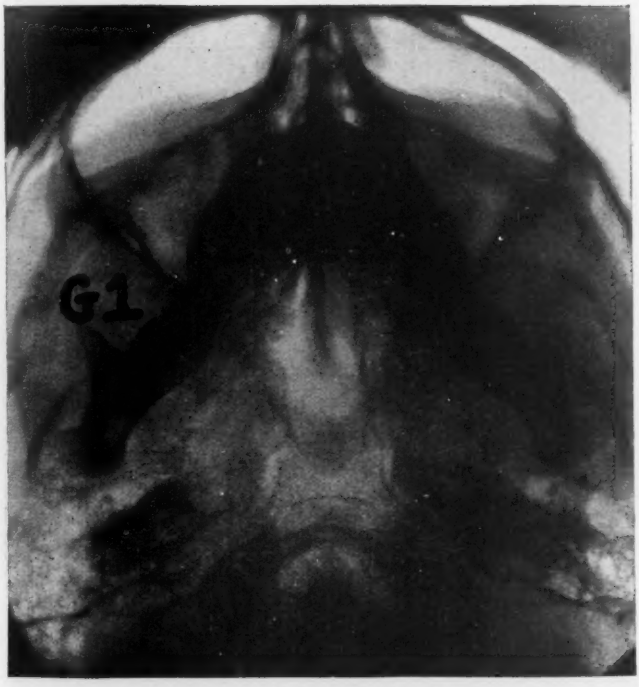
E3 shows the sinuses of the same patient as taken in the author's position No. 2. Antrums and sphenoidal sinuses are visible. The foramen spinosum, the foramen ovale, and the carotid canal are easily visualized.



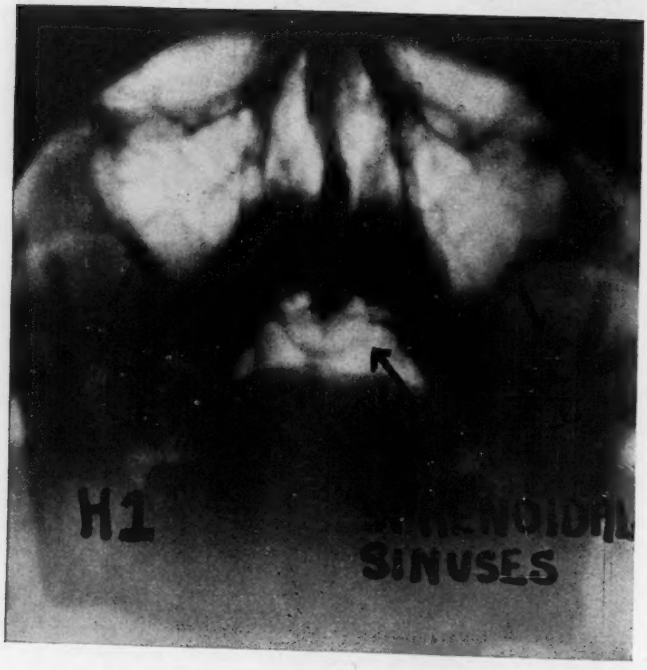
F1 shows the sinuses of a patient in Watter's position. The petrous portion of the temporal bone is seen to cloud the lower part of both antrums. The frontal sinuses are absent in this patient.

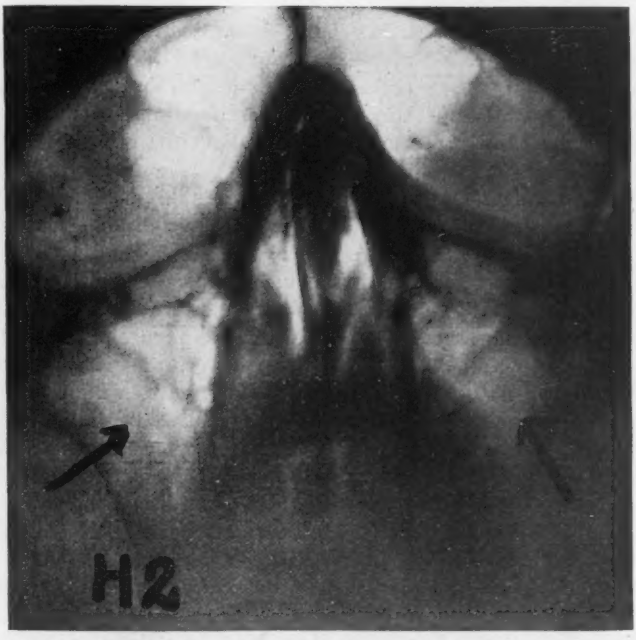


F2 shows the sinuses of the same patient in the author's position No. 1. The petrous portion in this case lies below the antrums and can thus can never cloud the lower portion of the antrums.



G1 shows the sinuses of a patient in the author's position No. 2. The antrums and the sphenoidal sinuses are seen. Besides, the mastoid cells on either side and both petrosal bones are seen. One can also visualize the foramen spinosum, the foramen ovale, and the carotid canal.


H1
SINUSES



H1 shows the sinuses of a patient in the author's position No. 1. The sphenoidal sinuses are readily seen. H2 shows the sinuses of the same patient in Water's position. In this case the antrums are clearer in the author's position than in Water's position.

OPHTHALMIC MIGRAINE; CONTROLLABLE THROUGH THE NASAL (SPHENOPALATINE-MECKEL'S) GANGLION; REPORT OF A CASE WITH UNUSUAL FEATURES.*

DR. GREENFIELD SLUDER, St. Louis.

Ophthalmic migraine has been a baffling syndrome from almost the beginning of medical literature. Discussions and theories about it are probably as numerous and extensive as those attending almost any ailment described at present. The attack begins with scotoma with or without the colored fortification margin. Sometimes the attack begins with hemianopia, or scotoma and hemianopia may alternate; this is followed in a varying length of time by headache, which, in my observation, is usually a "lower half" headache.[1] After the headache has lasted a varying length of time nausea follows with or without vomiting. This is succeeded by aphasia and this again by hemiparesis, sometimes hemiplegia. Such is the picture of complete ophthalmic migraine. The attacks, however, are rarely complete; most of them stopping with vomiting. Aphasia and hemiparesis are usually omitted. The attacks usually begin in childhood or adolescence and often there is a story of such attack in the patient's antecedents. The report of this case is made for what it may be worth without any desire to discuss at present the numerous theories that have been advanced to explain its mechanism.

Mrs. L. B. consulted me April 15, 1920. At that time she was sixty-three years of age. She gave a clear story of three types of pain in her head. At the age of twelve began clearly ophthalmic migraine in which aphasia and hemiparesis were omitted. The attacks occurred usually more than once a week, involving the right or left side irregularly. Scotoma, headache and nausea were severe at the time of our meeting. She stated that fourteen years prior to this began a typical tic-douloureux, i. e., major neuralgia of trigeminus, second and third divisions, left side, for which Dr. Harvey Cushing did a perfect Gasserian ganglionectomy with complete relief of the tic-douloureux. Four

*From the Laryngological Department, Washington University School of Medicine, St. Louis.

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years ago she developed a cyclic recurrent headache which she carefully explained was not at all the same as the migraine and could be relieved by abdominal exercises, which may be termed a "vulgar" migraine. Upon inquiry of Dr. R. W. Mills, who sent the patient to me, I learned that she had a non-malignant obstruction at the sigmo-rectal junction and that the abdominal exercises were directed to secure the passage of the bowel contents beyond this obstruction. Dr. Cushing's ganglionectomy was ideal in every respect. The result stands today perfect but the ophthalmic migraine and the vulgar migraine have continued unchanged. Having had success with ophthalmic migraine by nasal treatment I made the effort to discover some nasal pathology in her case. After a protracted, painstaking and satisfactory investigation extending over three months time I concluded that her nose was normal in every respect. I had much difficulty in getting to see her in the attack of ophthalmic migraine but finally proved that it was controllable through the nasal ganglion by cocainization. December 11 and 30, 1920, I injected the ganglia, left and right, using 95 per cent alcohol with 5 per cent phenol, operating through the nose. The reaction from the injection required two months before completion during which time she suffered slight pain referable to that procedure. In this interval, however, the ophthalmic migraine began to decrease in severity with longer intervals and finally ceased. She did, however, during this time, have a scotoma without headache or nausea at intervals of approximately six weeks. The scotoma continues now to the present to appear irregularly at considerable intervals (two or three months). (I have mentioned recurrent scotoma as an isolated phenomena of ophthalmic migraine.[2]). She enjoyed freedom from the migraine attacks until June, 1923, when she suffered a severe coryza which caused the attack (Scotoma headache and nausea) to return in moderate severity. The coryza ran the ordinary course with recovery in a little less than three weeks. The migraine continued for nearly three months and ceased without treatment of her nose. Since this time she has continued to enjoy freedom from ophthalmic migraine. The vulgar migraine (digestive headache) continues unchanged.

This case is at least rare (and to me interesting) inasmuch as the trigeminus of the left side is removed from all part in the causation of the ophthalmic migraine, and still the pain sensation during the attack is felt in the region supplied by that nerve.

This case is also very interesting to me from the fact that no recognizable nasal lesion existed. In all other cases of ophthalmic migraine that I have succeeded in helping there was either a hyperplastic, post-ethmoidal-sphenoidal lesion, in which relief was gotten by cocainization of the nasal ganglion; or a sphenoidal lesion with or without pus.

It is at present impossible to identify the starting point of the method or means from which or by which the attack was started in this case. The trigeminus was eliminated on the left side but the pain was felt in its distribution (first and second divisions) quite the same. It is also difficult to conceive of the deep sensibility of the seventh nerve (Loyal E. Davis[3]), as playing a part. Speculation leads one to believe that there are qualities or properties in the involutary nervous system (which becomes superficial in the nasal ganglion) that explain the attack and that what happens in this treatment is a block or brake, more or less complete, in its transmission tract whereby relief is obtained.

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1. SLUDER, GREENFIELD: Headaches and Eye Disorders of Nasal Origin, 1918. C. V. Mosby Co., St. Louis.

2. SLUDER, GREENFIELD: "Lower Half Headache (Neuralgic) of Nasal Origin. Glossodynia, Otalgia, Nausea Paraglossa, Vertigo, Scotoma, Photophobia, Rhinorrhea and Asthma, as Isolated Related Phenomena." *Journ. A. M. A.*, Dec. 2, 1922.

3. DAVIS, LOYAL, E.: The Deep Sensibility of the Face. Archiv. of Neurol. and Psychiatry, March, 1923.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

February 8, 1924.

Continued from page 496, June issue.

DR. EAGLETON expressed his gratification at being invited to discuss Dr. Shambaugh's paper, although he hardly felt qualified to do so. He had had it in mind to say a few words about the development of the sense of hearing, but after this wonderful demonstration there was no time to say it. Some years ago in helping to prepare a program for the American Otological Society, one of Dr. Shambaugh's papers was put on the program, and the Council of the American Otological Society then realized that there was not in America any research workers outside of Dr. Shambaugh. It was a wonderful thing for a man to devote for fifteen years of his life half of every morning and the whole of the holidays in doing something that will last forever. That is the way medicine has been built up—by someone doing something that lasts—not simply doing a mastoid, etc. The wonderful thing here though, is that Dr. Shambaugh combines both, the mastoid work and research. About two months ago he had been in Chicago and Dr. Shambaugh took him through his clinic, which greatly impressed him. If anyone wants to know how to run a teaching institution of Otology, he should go to Chicago, for there Dr. Shambaugh teaches his students the foundations of the pathology and anatomy of otology. He does not allow his men to go to the operating room until they have mastered these subjects. He is insistent on an idealism that we must all accept—that the successful operator is the man who knows—long before he applies his technique—the anatomical and physiological basis and when it should be done. There is too little of that in this country.

DR. SHAMBAUGH, in closing, expressed his appreciation of the comments that had been made on this work. What Dr. Eagleton said is only in part correct. Research work along anatomical lines in this special field has not been very extensive in this country but there have been a great many valuable research contributions which have a more practical clinical bearing than have been made by Americans. He stated that he had no explanation as to just how the glands of the sulcus spiralis externis perform their function; he had never made a close study of the glandular action. He stated that the hair cell, of course, is the structure where transference of physical impulses from sound waves to nerve impulses takes place. This is the real end organ. One thing which all the end organs in the labyrinth have in common is that all are stimulated by physical reactions.

SECTION ON OTOLOGY.

March 14, 1924.

The Relation of Otology to Neurology. Dr. J. Gordon Wilson, Chicago. (By Invitation.)

DISCUSSION.

DR. NEY: I have been greatly interested in Dr. Wilson's paper. From an experimental standpoint it is extremely valuable and interestitng. Vestibular examinations for differential diagnosis in neuro-surgery have been generally disappointing: In tumors of the auditory nerve it is quite possible to get a lesion involving some portions of the nerve to a greater extent than others. When one considers the auditory or any other nerve trunk, it must be considered as being composed of bundles or

groups of nerve fibres, and a lesion may involve only a portion of the trunk. In the vestibular nerve, reactions may be lost only in those bundles which innervate one canal and which terminate centrally in an individual path, in which case the vestibular reactions alone might indicate a central lesion.

I have been very much interested in Dr. Eagleton's reports concerning the vestibular findings in intracranial lesions associated with increased pressure. In my clinic we have been able to verify his conclusions to the effect that early in a case of increased intracranial pressure there is often a diminution or loss of response to stimulation of the vertical canals. In many instances of increased intracranial pressure we find not only marked changes in the differential reaction of the various canals, but also occasionally a marked diminution or total absence of all the vestibular reactions to both caloric and turning tests.

We cannot rely on the examinations of the fundus in determining increased intracranial pressure, though the fundus often suggests an increased pressure by congestion of the retinal veins or varying degrees of papillary oedema: we often find that marked intracranial pressure produces few if any changes in the fundi. Our determination of intracranial pressure is made during ventricular estimation, when a manometer is attached to a needle passed into the lateral ventricle, and the degree of pressure accurately measured. We find frequently that a differential disturbance of vestibular reactions will indicate an increased intracranial pressure when the fundi are apparently normal. Inasmuch as most neuro-surgical lesions are associated with increased intracranial pressure, and that increased intracranial pressure alone will produce marked disturbance in the reaction of the various canals, we must conclude that vestibular reactions have no dependable localizing value in lesions associated with increased pressure, and that conclusions should not be drawn from vestibular reactions insofar as localization is concerned until the intraventricular pressure has been definitely measured (which must be done under local anesthesia, as the straining associated with general anesthesia will frequently produce a rise of from 10 to 20 mms. Hg.).

Dr. Pike refers to the respiratory disturbances caused by a blood clot in the fourth ventricle. I would like to know his method of irrigating the fourth ventricle for the removal of blood clots and the solution used in the irrigation.

DR. EAGLETON said that this work by Dr. Wilson and Dr. Pike was undoubtedly the finest thing that had been brought before the Section on this subject. The only way we will ever get anything with the vestibular tests is on animals and by clinical examination of the patient, and, in case of death, by post-mortem examination, both gross and microscopic, thus following up the case to find the cause. All the postulates that have been made are simply theories and do not rest on any facts, and they have done a great deal of harm. There are reports of localizations of tumors in all parts of the brain, by the vestibular tests, in the literature. Dr. Eagleton said he had spent the best part of a voyage to Europe to try to understand the mental process by which such localization was reached. There is no possibility of explaining it on any theory whatever. A time will come when the profession can understand and use the vestibular reactions for localization. He believes thoroughly in the vestibular reactions and considers them the greatest addition neurology has had since the discovery of the ophthalmoscope; Dr. Eagleton said that if he had to throw away one or the other he would rather throw away the ophthalmoscope than the turning chair and caloric, for one can find as much or more with the turning chair than from the ophthalmoscope in neurology. From the caloric we may say, here is intracranial pressure, but that is all. We can diagnose an increased intracranial pressure of the posterior fossa by vestibular reaction; even when there is no evidence whatever of papilloedema. Dr. Pike stated that an instrument has been devised so that you can photograph and thus tell the direction of the nystagmus. The ophthalmoscope will do this if you have your

patient caloriced or turned in a dark room and then looked at by the indirect method of the ophthalmoscope; you can tell whether the nystagmus is right or left for you have a magnification of many diameters and you can tell in a moment the direction of the quick or slow component. Dr. Eogleton said he would recommend that to those who had not tried it.

Referring to the nystagmus of the tempero-sphenoidal lobe, Dr. Eagleton said that Dr. Wilson's paper was dealing entirely with acute traumatic lesions. The same thing happens in abscess of the brain. If you have an abscess of the temporo-sphenoidal lobe on one side, there is loss of the quick component, and at this time you may have a slow deviation without any quick return. The quick component has been destroyed in the tempero-sphenoidal lobe by the abscess. This is of marked value, but as soon as compensation sets in, it is lost.

As to the cause of nystagmus in general, Dr. Eagleton said he has a theory, in a paper which has never been published—and perhaps may not be worth publishing—that this thing can be explained in a rather logical way; if you take a lower animal and turn it, you don't produce a nystagmus, but a lateral deviation; on top of this lateral deviation there is built in the higher animal a nystagmus, and we are never able to produce that until we get to the mammals, and it has to be a rather high mammal before one can produce a decent nystagmus. The explanation seems to be that it has come up in development; it had the fundamental deviation due to disturbance, etc., the eyes of the animal gradually came together. The higher they become, the nearer the approach to binocular vision which man alone possesses. You can produce a nystagmus in man which you cannot produce in any animal; it is only in the mammals which come up toward man that you have any nystagmus at all. Man is the only creature that has a claim to binocular vision. It is the attainment of binocular vision which gives the nystagmus from lateral deviation.

Now let us throw out of our calculations all this idea that we can localize a chronic lesion of the cerebellum from a vertigo or nystagmus. There is a definite nystagmus from acute cerebellar lesions, but it lasts only a short time. Holmes found that soldiers who had been shot in the cerebellum had a distinct nystagmus, but it lasts for only a few days. Thus spontaneous nystagmus is most marked from a "fixed point". That is definite, and distinct type of nystagmus. This is the cerebellar nystagmus, but it lasts for only a few days.

DR. E. M. JOSEPHSON: Dr. Wilson's paper points out the apparent difference between the ocular reactions to vestibular stimulation in humans and the lower vertebrate forms. Clinical observation, however, indicates that the difference is apparent rather than real. It must be realized that nystagmus is not a normal form of reaction to vestibular stimulation but is a sign of vestibular irritation, a response to intensive stimulation. The ocular responses to vestibular stimulation of a normal, or minimal type are two; first, the compensatory eye rolling, first extensively studied by Barany; and second, a reaction which has recently come to light and has been named the heterophoric reaction. Both of these reactions may be elicited by minimal stimulation, viz., if the patient be turned through a fraction, one, or two turns. These reactions are of the same type as the reactions seen in the lower vertebrate forms —they consist of ocular deviations rather than nystagmus movements.

The compensatory eye rolling movements are best studied by turning the patient through a fraction, one or two turns, with eyes rotated in the same direction as the turning. A few adults can with effort hold their eyes in the same position throughout the period of turning; if the vestibular apparatus of the patient be normal, the natural tendency of the eyes is to roll in the direction opposite that of turning, and the average patient when attempting to hold his eyes in the original position, rotation in the same direction as that of turning, will execute a pendulum-like eye rolling toward the mid-line and back to the original position. The movement of return to the original position is in these

cases obviously a volitional act, and, with all other volitional acts, can be assumed to be due to a cerebral impulse. In children, who possess weaker volitional powers, it will usually be found that the eyes are rolled at the end of rotation from the original position and turned to the side opposite the direction of rotation. In deaf-mutes with "dead" vestibular apparatus, eye rolling can seldom be elicited.

Heterophoric reactions differ from the eye rolling reactions in that they may be monocular. These effects are studied as follows: The eyes of the patient are first examined to determine what be the state of balance of the eye muscles. The patient is then turned through a fraction, one or two turns, and the eye muscle balance then re-examined. If the vestibular apparatus be normal, it will be found that a change in the muscular balance has taken place. The change is first observable on the side opposite to the direction of turning, and with minimal stimulation is monocular. When the stimulus is increased, the change is observable on both sides. The nature of heterophoria induced varies with the original condition of the eyes. No rule has yet been discerned or formulated; further studies in this direction are under way.

The attribution of the quick component of nystagmus to cerebral influence, the explanation offered therefor in Dr. Wilson's paper, is supported by various clinical data. As we have above noted, the response of the eye to minimal vestibular stimulation is a deviation of one or both eyes, corresponding to the reactions found in lower vertebrate forms. When the vestibule is further stimulated, or, inasmuch as such stimulation is beyond the range of the normal, when the vestibular apparatus is irritated, nystagmus and vertigo are elicited.

Light is thrown on the nature of nystagmus reactions by the study of the compensatory eye rolling above noted. The pendulum-like eye rolling which is elicited by the attempt of the patient to fix his gaze in the direction of turning constitutes the elemental form of nystagmoid reaction, and is regarded by some observers as a true nystagmus. In this case the return movement is obviously volitional and of cerebral origin. This is rendered more obvious by the fact that if the eyes be rotated in the direction opposite that of turning, the volitional and vestibular impulses coincide and no eye oscillations occur.

In the after-nystagmus following maximal stimulation of the vestibular apparatus, the same fact is noted, i. e., the nystagmus is most marked when the eyes are rotated in direction in which the volitional and vestibular impulses do not coincide. This again indicates that conscious or subconscious volitional effort may underlie the quick component of nystagmus.

In humans with extensive cerebral lesions, the rapid element of nystagmus may be entirely absent. On the other hand it has been observed by Henri in frogs, and, as mentioned by Dr. Wilson, by Tredelenberg and Kuhn in tortoises, that any improvement which occurs in the animals in the way of return of normal posture, is promptly destroyed by decerebration of the animal. Hence it may be surmised that even in lower vertebrate forms, the return to the normal resting position, which corresponds to the quick element of nystagmus, is cerebral in origin. The effect of anesthesia was mentioned in the paper.

Loeb's principle of algebraic summation of optical and vestibular stimuli, first demonstrated on a species of toad implies a prosencephalic involvement in reactions to vestibular stimuli. Light is thrown on the nature of this type of summation in the human species by an interesting reaction first noted by Barany but discounted by him because he regarded it as a purely cerebral phenomenon. This reaction has recently been proven to involve the vestibular apparatus, insofar as it is absent in cases with lesions of the vestibule, on the side of the lesion. It has therefore been named the oculo-vestibular past-pointing reflex. It consists of past-pointing in normal individuals when the eyes are closed and rotated in the direction of the pointing arm. This reaction is the converse of the vestibulo-ocular reflex and indicates the presence of both afferent and efferent fibres in the vestibulo-ocular tracts.

The possibility of altering the responses of the vestibular apparatus in the human by altering the position in which the eyes are held during rotation points to the summation of impulses. If the patient be ordered to rotate his eyes upward and outward in the direction of turning and to hold or try to hold his eyes as fixed as possible in that direction during the ten turns of rotation, it will be found that there has been elicited an eye rolling or nystagmus which is most marked when the patient holds his eyes in the position which he has been ordered to assume during the turning. Nystagmus may be entirely absent when the patient looks in the direction opposite to that of turning or straight ahead. This is a reversal of the findings ordinarily met with when the patient is rotated with eyes closed. The reaction to vestibular stimuli is alterable by changing the position assumed by the eyes during turning. The site of summation of the impulses* is not yet determined.

*Ocular and vestibular.

DR. PIKE, closing the discussion said that Dr. Wilson had some clinical cases, and in the section on cerebellar nystagmus he had a report on the first patient who was carefully examined after a shrapnel wound of the cerebellum. Dr. Wilson was in the British Army Medical Service, and had not been there long when this fellow was brought in. The shrapnel was dug out, so that there was a pretty good idea of what had happened in the cerebellum. The report was rather too long to read, but would be published. There were also other cases of the same kind.

In the matter of intracranial pressure, no one could tell exactly what it was going to do or what it was going to effect. Some years ago Dr. Wilson and he had pointed out this difference between clinical and experimental cases. Since all signs fail in dry weather, pretty nearly everything fails in intracranial pressure. One has to have something pretty definite and know where the primary thing is. The whole vestibular mechanism is so complex, that for a time he gave up trying to analyze it on the present basis and started on some simpler system. The respiratory system was a little simpler, and had some advantages that nystagmus does not have. He wanted to get some system for analysis, and it does not make much difference what system is taken first.

In the matter of experimental work, there is the time factor to consider. It is necessary not only to do an experiment, but it is important to make these experiments and then allow a long time to elapse for some of the slow processes to take place, to approach these processes of compensation which do not take place in the nervous system. They do not come on suddenly. That means keeping the animals alive for a long time, and this is a difficult matter. Some have been kept for two years. One soon gets a whole boarding house full of them, so that the universities are apt to consider them as luxuries, and the anti-vivisectionists consider them as something worse. The work is slowed and progress becomes difficult.

Dr. Ney had inquired about the method of washing out the fourth ventricle. In going in there are two routes by which we approach the cerebellum in some animals; one by bending the head forward and exposing the foramen magnum and then going in; one can lift up the cerebellum. That has been done by others. Fraser lifted up the cerebellum and went into the fourth ventricle, blocking the aqueduct to produce hyprocephalus. So long as one does not do anything else he does not have much hemorrhage, but if one sections the cerebellar peduncles or gets into the cerebellar lobes hemorrhage is apt to occur.

Dr. Pike said his own way was to pour in some water—very warm water that had been boiled—and wash out the clot. The clots were usually not very adhesive, and with fine forceps could be removed almost entire. In going through the fourth ventricle and loosening the adhesions, he advises wrapping cotton about the forceps to avoid injury to the nemosis tissue. The fourth ventricle is an exceedingly sensitive region.

He was much disappointed that Dr. Wilson was not able to hear this discussion and take part in it. The statement made some years ago was

that not only would some of the current ideas of localization of central lesion by ear tests prove disappointing—but they had already proved disappointing—and he was very much pleased to hear some confirmation of that statement this evening, for a while "we" were very lonesome.

It took some time for the idea that perhaps there was some cerebral influence upon the mechanism of nystagmus to gain recognition. He had noticed that in some cases of central organic lesions of the nervous system, diagnosed clinically as lesions of the corpus striatum and characterized more by disturbance of movement than of attitude, nystagmus was increased on looking to one side or the other when the patient was lying down. Dr. Pike said he had no idea what the explanation was. While there may not be any real nystagmus on sitting up, one frequently does get marked nystagmus from a patient lying flat. If one has a nystagmus sitting up, it is usually increased when lying down. These lesions would be diagnosed on the basis of the older views as being in the posterior fossa. In one case of temporal abscess, observed with a friend, from some peculiarities of the nystagmus from caloric stimulation he had said there was something in the temporal fossa, but no one believed him until the post mortem. The abscess was found in the temporal lobe.

The clinician looks at these things from one point of view and the experimentalists from another, and unless one is pretty careful he is likely to miss the common ground of understanding and simply increase confusion. There is much that we do not know and possibly never will know, but there is also much that we do know. Everyone thinks it is absolutely necessary to know the anatomy of the nervous system, or of the ear, or whatever he is studying—seeing the structure in three dimensions; sometimes apparently one can draw conclusions from the fourth. But how many have found it equally necessary to watch experimental animals? Very few. It is difficult for an experimentalist to talk to a clinical audience. A clinical man can talk to them, but the difficulty of describing the deportment of experimental animals to those who had not studied them carefully, was greater than the difficulty of describing anatomical preparations or clinical.

Dr. Pike said that he trusted he had not added too much in the way of confusion but rather that he had added somewhat to their understanding.

SECTION ON OTOLOGY.

April 11, 1924.

Secondary Mastoiditis Complicated with Localized Meningitis, Bilateral Paralysis of the External Recti Muscles of the Eye, Ptosis of the Eyelid, Choked Disc, Sinus Thrombosis, Recovery. Dr. James J. King.

This very interesting case was admitted to the French Hospital on December 8, 1923, with a temperature of 104°. She had had a mastoid operation five or six years previously in Central America. On examination, she showed tenderness over the right mastoid and a swelling which looked like a subperiosteal abscess. Immediate operation was advised, and was performed as soon as permission was secured. At that time she had meningeal symptoms and very interesting eye symptoms; she had paralysis of both external recti and ptosis of the right lid, a mastoiditis on the right side, and a slight swelling of the face around the lips. The eye examination showed a papillo-edema with swelling, more in the left than in the right eye; she had double vision when she looked outside of the range of this motion of the eye.

At operation a very small mastoid cavity was found, with the sinus against the posterior wall. The sinus was completely thrombosed and the meninges were injected and red. The jugular was ligated and the

sinus was obliterated in the mastoid cavity almost half way back to the torcula. The sinus was thrombosed throughout the mastoid wound. A very large area of the dura was left uncovered; the wound was left open and dressed with iodoform gauze.

The patient was quite sick and had a very stormy time, the temperature ranging from 103 to 105 or 106 for several days. Brain abscess was suspected, although at all times her co-ordination was very good. The wound was treated with Carel-Dakin solution. The brain is still uncovered and there are superficial vessels which bleed very easily, and in the last few days it has been discharging. The patient is now at home and comes to the office for dressing as needed; her tongue has cleared up and she is practically well.

Brain Abscess and Encephalitis: Differential Diagnosis. Dr. Alfred Kahn.

On March 5, 1923, I was called to see Mrs. E. H.; she gave a history of otitis media and having had influenza. She was not over the attack at that time. Her temperature was 100.5. On examination she was found suffering from an acute otitis media (a bulging ear-drum) in the right ear. A paracentesis was performed (right ear) and a lot of pus was evacuated under tension. She showed signs of mastoid involvement and was sent to the hospital. (The left ear was normal.) A consultation was held that evening, and the following afternoon a mastoid operation was performed; her temperature at the time being 103. It was a hemorrhagic type of mastoid, and bled excessively during the operation, so that it could hardly be controlled by sponging and the operation was practically done in a bloody field. A small area of the dura was exposed, and the sinus was opened with profuse bleeding above and below. The sinus was packed with the usual technique and the patient was returned to bed.

The temperature immediately dropped to 100, but the next day it suddenly rose to 103.5. Then occasionally, off and on, the patient would talk out of her head, saying a sentence without meaning, and then stop and talk normally. Three or four times a day she would make these irrational remarks. The nurse called attention to this point, but it was thought to be a post-operative disturbance and that she was more or less hysterical. This continued for several days, however, and brain involvement began to be suspected; although it was difficult to believe this, since before the operation there was no history suggesting it, and while the dura was exposed during the operation it certainly was not injured. Of course, the sinus was exposed, and the patient was carefully watched for signs of jugular involvement or involvement of the lateral sinus; but there was no chill or sweating, and the examination of the blood showed nothing.

As the mental condition could not be definitely explained a neurologist was called in consultation. He examined the patient and found the abdominal and other reflexes absent. He suspected encephalitis. A spinal puncture was done and other examinations were made to determine whether there was encephalitis. There was an unquestionable meningitis but it did not look like the usual type found after mastoid operations. In taking the spinal fluid we had in mind the determination of a simple meningitis and the determination of encephalitis. If the spinal fluid had shown cells, or had shown the presence of pus germs a decompression would have been indicated. It did not show these elements; but it did respond to the polyofixation test and encephalitis was therefore diagnosed. There, however, was no definite sign of a brain abscess. The patient at this time showed a partial Kernig and a partial Babinsky in the left leg. The right leg was slightly involved. The patient would look excellent for a few days and then suddenly lapse into somnolence. This continued for a little over a month, until, finally on April 7, the patient died, the diagnosis still being in doubt, or rather the impression being that it was a pure case of encephalitis.

An autopsy was performed, and a very large tempero-sphenoidal abscess was found.

Looking backward it is easy to read the signs differently. Doubtless, all you men present have seen brains probed for abscess and none found, and, then after death (in other cases), found abscess at autopsy, that easily could have been reached by operation. How serious it is to go into a brain without definite signs. I do not know of any more difficult problem in surgery than the determination of right-sided-tempero-sphenoidal abscess. How sad it is to probe a healthy brain through an infected area and to have the subject die from the probing; and yet how disappointing to have a patient die from an abscess easy to reach. What can I draw from this case? To what extent should the otologist rely on the neurologist. The state of mind of the two men are so different. The neurologist is entirely in a medical state of mind. The otologist is entirely in a surgical state of mind. A splitting of hairs between them may decide life or death. In this case the otologist was interested in a brain lesion. Is it a pure case of meningitis. Is it a possible brain abscess? Can surgery do anything? The neurologist is not able to answer these questions any more than the otologist. All he can do is to give him data to answer these questions for himself. He is not willing to make a positive diagnosis of brain abscess. The otologist should bear this in mind. And you cannot perhaps blame the neurologist. He has a very much wider knowledge of the brain than the otologist but even he is not able to make a sure diagnosis of right-sided brain abscess, and a decision of this type, gentlemen, is a serious matter. Looking back, up to the operation, this patient was all right. Immediately after the operation she presented the first sign of mental involvement. Of course, one case is not enough to go by and one cannot lay down definite rules but the juxtaposition of a mastoid operation and brain symptoms should bring into mind a consideration of brain abscess. Another sign is this: We all know that brain tension is apt to be associated with a slow pulse and a slow respiration. This is especially so in cerebellar abscess. You can notice by the chart in this case that while the temperature was vibrating between 100 and 104, that the respiration was hardly at all affected from the normal. It is very important to watch the pulse and respiration where there is any question of brain tension. The abscess was unusually large and it would have been extremely easy to enter. The autopsy showed the dura in perfect condition and I am sure that the infection did not enter through the middle fossae following the operation.

Another question: Why did this abscess present symptoms immediately after the mastoid operation. Dr. Eagleton spoke about this some years ago. He had noted brain abscess often followed the radical mastoid operation. I think that where you have an enclosed abscess, that when the brain is exposed (a decompression) that there is a reaction on the part of nature in relation to the pressure, to enable the abscess to break through.

Here we have an encephalitis, a very difficult condition to diagnose, complicated by a tempero-sphenoidal abscess.

DISCUSSION.

DR. M. NEUSTAEDTER, referring to Dr. Kahn's remarks that he did not wish to criticize, said that he was perfectly willing to be criticized, that he was not ashamed to hear the truth and to acknowledge mistakes. No one is infallible or can diagnose correctly 100 per cent.

He saw the case in consultation with Dr. Kahn, March 21, 1923. There was nothing in her family history or previous history that would have any bearing on her present condition. The present story was that on March 2 she had grippe and March 5, a radical mastoid was done. Within 24 hours after the operation her temperature shot up to 104 and continued for two weeks dropping down to 102-101. Most of the time she was confused and did not recognize her people. At the time of the examination she was deeply somnolent and aroused with difficulty. Her

pupils were irregular and unequal, the left being larger, sluggish to light and prompt to accommodation. The ocular fundi were negative. All abdominals were absent, patellars brisk, there was an unsustained clonus on both sides and plantar flexion on both sides to all methods. Kernig was positive and the neck was markedly rigid. Sensory phenomena could not be tested on account of the lack of co-operation on the patient. There was a left hemiparesis and a left ocular ptosis. Her temperature was then 102.2. The blood picture was 4,640,000 red cells and 9,600 white cells, 78 per cent polyps, 16 per cent lymphocytes. 20 cc. of spinal fluid under increased pressure were withdrawn. It contained a few lymphocytes globulin+++, Wassermann negative, lange 2223443210, polio fixation +++, no bacteria on smear, culture negative. A guinea pig was injected and showed no lesions.

Two days later she showed marked rigidity of the neck, completely stiff pupils to light and was profoundly lethargic and markedly confused when awakened. There seemed to be now a tendency to extension toe on the left foot.

The question arose as to whether we were dealing with a brain abscess or lethargic encephalitis. A stiff pupil, absence of all abdominals, no papiledema, ptosis on the left side, dilated pupil on the left side, the sudden rise of temperature to 104 within 24 hours after the operation, the lethargy, the history of grippe and the +++ positive polio fixation in the spinal fluid conclusively argued against an abscess.

During the last six years I have in association with Drs. Banzhaf, Larkin and Hala worked out a specific antigen that fixes complement in spinal fluid of cases of poliomyelitis and encephalitis. The latest report on this work you will find in the *New York State Med. Jour.* of the January, 1923 issue. A very large amount of spinal fluids of frank and suspected cases was examined and controlled by hundreds of fluids of pathological conditions other than encephalitis and of normal humans. The results were most encouraging. The chemistry and cytology of the spinal fluid in encephalitis per se give no clue that would be a criterion of the disease. You will find in any inflammatory state of the meninges irrespective of the etiology a higher cell count and an altered chemistry. As a matter of fact the cell count in encephalitis may be normal. The positive fixation with our antigen is the criterion and we went by it. In all spinal fluids with brain abscess the fixation was invariably negative.

Having decided that we were dealing with lethargic encephalitis our antipoliomyelitis horse serum was administered intravenously in 20 cc. doses at 24-hour intervals. Four injections in all were given with very good results. The lethargy began to clear up, the temperature went down, the mental symptoms disappeared and her left paresis diminished. Her improvement became so marked that we now contemplated removing her to her home.

Two weeks after the last injection of the serum her condition turned worse. The paralysis increased, the rigidity of the neck became more marked, double chocked discs with retinal hemorrhages appeared, the right pupil was now the larger of the two. At this point I suggested to Dr. Kahn, if he remembers, that there might after all be a superimposed abscess. Her condition grew worse and she finally expired. The autopsy, as reported, revealed a brain abscess. The microscopic and histologic examination did not reveal an encephalitic lesion.

It does seem that the encephalitis must have completely receded. I am of the opinion that we at first had encephalitis and I am supported in the view by another case that came autopsy in the King's County Hospital. A patient came in moribund. The post-mortem revealed both brain abscess and encephalitis. Ante-mortem his spinal fluid gave a ++ positive polio fixation. I am sorry that there is no microscope at hand to show the characteristic lesions of encephalitis in the sections taken from the various parts of the brain.

Of course, it is quite impossible to lay down a definite rule which would positively guide us with any degree of certainty in the early

diagnosis of brain abscess. But I feel that it is rare for a brain abscess to begin within 24 hours after a radical mastoid operation with a sudden rise of temperature to 104. Brain abscess temperatures hover around 99-100, and the onset is gradual and insidious. The case is of great interest and were it not for the other case that was so clear cut as to the fixation, I would seriously doubt the value of our complement fixation. I invite the most searching criticism.

DR. HAYS said it might be some consolation to Dr. Kahn to know that he himself had had two cases that showed no symptoms of an intracranial condition until after the mastoid operation. In each instance a latent brain abscess was present which lit up acutely when an infection occurred. One of the patients was the wife of a physician. The operation revealed a very vascular mastoid; afterward she developed very peculiar symptoms and died three weeks later. Autopsy revealed a large tempero sphenoidal abscess of which there had been no indication and which was not directly connected with the mastoid infection. In another case the patient had been suffering from severe headaches for over twenty years. The man had been examined a number of times but no intracranial lesion was found. He finally developed an acute sphenoiditis, a diagnosis which was confirmed. Much against his will, Dr. Hays had to operate. When the sphenoid was opened a probe was inserted which went through a dehisence in the posterior wall and entered a large abscess cavity. The patient died of meningitis two days later. This case was not confirmed by autopsy. In other words, it is possible to have a latent process in the brain after giving no symptoms until an acute infection of the nasal sinuses or the ear takes place.

DR. LEDERMAN said that these brain contributions were too serious and important to pass by hurriedly without due consideration and discussion. He had operated on a young woman with a history of chronic suppuration and an acute mastoid. She had gone on for a long time and had refused surgical treatment. She was finally operated upon and extensive destruction of the mastoid process was found, with a perforation of the posterior canal wall, but made a good recovery with cessation of the aural discharge without atresia of the canal. Seven months after the operation she returned, and the only symptom she noticed was a little swelling on the upper line of the mastoid incision which was soft, not painful, and showed no signs of inflammatory condition but only what seemed to be a little cyst. The question arose whether it was a sebaceous cyst of the scalp or whether it was associated with the former operation. She had no mental or general symptoms and no elevation of temperature. Examination was advised to determine the possibility of its being a hernia cerebri, and she was sent to a prominent otologist who said he could not arrive at a definite opinion although it did not seem to him to be a hernia cerebri, and he agreed with the speaker that it should be explored, which advice the young woman refused. X-ray examination offered no assistance in the diagnosis. One month later, Dr. Lederman was called hurriedly to see this patient and found her unconscious, temperature 102½°, with rigidity of the neck, Kernig's symptom, dilated pupils, and had vomited several times. Meningitis existed and a cerebral abscess was suspected. The patient was rushed to the Lebanon Hospital for operation, with the expectation of finding a large local lesion. Much to his surprise, on opening up the swelling,the dura was found under the skin. The brain tissue was incised, but no pus could be detected. With the exploring needle, four different sites were punctured, and then an incision was made with a knife, but no pus was found.

She came out of the coma in forty-eight hours and her mental symptoms cleared up. A lumbar puncture was performed at the time of operation; the fluid was cloudy, truculent, came only under pressure, and showed a count of thirty cells. A diagnosis of meningitis with cerebral abscess was made, but no abscess was found; it was a cerebral hernia. There was not much pressure on the optic nerve as revealed by inflated examinations. The patient recovered sufficiently to get out of bed. Dr.

Elsberg examined her and at the time thought her symptoms suggested the presence of a cerebellar abscess. She had a little horizontal nystagmus. She improved sufficiently to leave Lebanon Hospital and was transferred to the Neurological Institute, where Dr. Elsberg explored the brain in four different directions, but found no pus. She then went home and gradually became worse. She passed away seven months after the brain exploration, but unfortunately no autopsy could be obtained. Symptoms of cavernous sinus thrombosis of the same side (left) were present at her death. Such cases show the difficulty in determining just where the lesion is located. The cerebral hernia was probably due to hydrops ventriculi, caused by a cerebral abscess not discovered.

In still another case, a secondary operation was done on a young child who showed distinct signs of meningitis following an aural infection. The child had been operated upon a year before at another hospital and after the second operation the entire condition cleared up after the removal of diseased bone in the region of the middle fossa.

It is remarkable how many complications some of these cases will stand and recover. In one case a girl of fifteen was sent to Lebanon hospital by her family physician for a medical condition, with a temperature of 103°. She was examined on the gynecological service and found to be three months pregnant. One day, following a chill, the temperature rose to 107°, and a suppuration of the middle ear was found, which she said she had existed for a long time. The usual blood examinations had been made but except for a pronounced leucocytosis and a marked increase in the polynuclear cells, nothing further was found. A diagnosis of sinus thrombosis was made, the vein was ligated, and the sinus exenterated. She began coughing quite frequently, and seven days after the operation she had a severe paroxysm of cough and expectorated an unusual quantity of foul-smelling pus. She had aborted on the third day following the regular ligation. In spite of a large pulmonary abscess, an alveolar abscess on the right side due to an infected gum, an extensive cerebral hernia and curettage of the middle ear for removal of granulation tissue, the young girl made an excellent though stormy recovery with good hearing.

We surely must accept any suggestions or advice from our neurological confreres that their experience may offer. Cerebral localization is an important aid in these serious cases and may lend considerable aid to the operator.

The Masking of Sound, Etc. Dr. E. P. Fowler.

(To appear in subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. WEGEL said that Dr. Fowler deserves a great deal of credit for the amount of work that he has done in the interest of pure scientific research. He has spent a great deal of his time, both in doing experimental work and in the study of the more abstract problems connected with the functioning of the ear, which it is to be hoped will at some time prove of service.

The natural question which arises in one's mind after hearing a paper of this kind is, What is the practical use of all such information and speculation? and it seems to me that an answer may be found by looking at the problem as a sort of prospective. This problem in common with many others of like nature may be regarded from the standpoint of about four phases. These are: 1. Speculation; 2. Experimentation; 3. Correlation of Experimental Results; 4. Practical Application.

There has been a great deal of speculation before any exact experimental work could be done on how the ear functions and advocates of the various theories have been at a loss to come to any substantial agreement, because there has not been enough experimental evidence to practically demonstrate the correctness of one view or another. A great deal of exact experimental work has been done during the last

few years on the various aspects of the functioning of the ear. In these might be mentioned measurements of absolute sensitivity or acuity, measurements of sensibility or the ability of the ear to detect small differences in both intensity and pitch, measurements of ability of the ear to interpret speech sounds with various definite frequency ranges eliminated and the amount that one kind of a sound may mask another sound of known amount. Many measurements have also been made on abnormal ears, as to their acuity and property of physically distorting sounds. A large amount of data has, of course, been available on the anatomy of the ear and this coupled together with the experimental data and a knowledge of theoretical dynamics, should serve as a sound basis for deducing the mechanism of the ear. Any theory which is able to account for all of the phenomena observed should be regarded tentatively as correct, and no theory which fails to account for one observed fact may be considered adequate. If a sufficiently large mass of data is available and a correlation between this and more abstract dynamical philosophy is accepted, there is a likelihood of obtaining a practical application to otology and other sciences.

The ear mechanism is so small and so inaccessible that it is not possible to make any direct observations on the minute stimuli employed in the nerve terminals within it during life. Attempts at such observations on preparations made after death may hardly be expected to produce results because of the fact that the resilience and other physical properties of the tissues undergo a considerable change. We are, therefore, left only the indirect method of attack; that is, a measurement of all characteristics possible and correlation of these things by means of the standard principles of dynamics.

My work has been largely connected with the study of normal ears from the standpoint of a telephone engineer. The purpose in this study has been to learn more about the functioning of the ear and of the voice so as to more intelligibly design telephone apparatus which will accommodate them more satisfactorily, and to improve the telephone service.

Dr. Fowler has talked at length on the way the ear appears to function in the light of recent data and I think I can add but a little on the details of the apparent working of the ear. The method of determining the position of the basilar membrane at which stimuli for sounds of various frequency take place is a very simple one. If you take two points of a pair of dividers and apply them simultaneously to the finger tips, it would be found that in order to detect them as being two separate points, it is necessary that they be more than about two millimeters apart. If the same experiment is done on the tip of the tongue, they need only be about one millimeter apart while on the back of the hand the figure is several centimeters. The basilar membrane is regarded as an extended region in which tactile nerves terminate and are more or less uniformly distributed along its length. If then a sound of a certain pitch produces a vibration of this membrane at a certain point and another pitch produces a vibration at a short distance from it, these pitches will be distinguished as different when the regions which they stimulated are far enough apart; that is, a distance analogous to the two millimeters on the finger tips. Making use of the frequency sensibility measurements then, which are nothing more than a measurement throughout the entire audible range of the smallest change in pitch distinguishable, and assuming that one of these increment of pitch changes corresponds to the stimulation of adjacent regions equally spaced on the basilar membrane, it will be easily seen with a knowledge of the length of this membrane, that the actual position at which each frequency affects it can be determined. Dynamical considerations show that the highest frequencies must be detected at the proximal end and the lowest near the helicotrema. By this method, it is found that a vibration of 1000 DV or approximately two octaves above middle C stimulates the nerve terminals in the mid point of the membrane. A frequency of about 300 DV or G above middle C, stimulates it at three-quarters of

the way up to the helicotrema and about 3,000 DV or G, three octaves and a fifth above middle C, affects it one-quarter of the way up.

The interpretation of the masking data may also be made a little plainer by means of the analogy of pressing the points of a pair of dividers in the fingers. When the two points are pressed so close together on the fingers as to produce about the same shape dent as the one point alone, especially if one of the points presses harder, the point pressing harder produces a sensation which may be said to mask that produced by the other. It is by means of a philosophy of this kind that we have been able to give tentative curves of the vibrating depression in the basilar membrane when agitated by pure sounds.

It is a well-known fact that the absolute sense of pitch is not very accurate. This means that the basilar membrane may not be stimulated at exactly the same point each time for the same sound. It is reasonable to expect variations in the characteristics of the membrane and other attached structures, such as the dilation of the little blood vessel underneath it as observed by Dr. Shambaugh. Such variations probably mean a variation in the actual position at which a certain pitch is sensed. Sounds are perceived as far as pitch is concerned relative to other sounds and the appreciation of music depends not on the absolute pitch or position on the membrane, but on the musical interval between it and other sounds of the melody or cord. Every one can distinguish large differences in pitch, but few can fix with any accuracy at all what the pitch of a note sounded is unless given in reference to another one. It may be well to note also in this connection that equal musical intervals are equally spaced along the membrane, except at its extremities where the sense of pitch is not well marked any way.

DR. GUTMAN sad it was impossible to discuss such a paper by merely listening to it; that it must be very carefully read and studied before discussing it intelligently. It was found that a person with a good musical ear could distinguish the difference between 1,100 and 1,101 vibrations, but how this could be applied to a musically untrained clinical case he could not understand.

Referring to the question of masking, Dr. Gutman said he would like to know whether a high frequency note could be masked as well with a low frequency note as a low frequency note with a high frequency note. He did not know whether this theory could have any application to cases of paracousis Willissi, where a person can hear better in the street with outside noises than in a quiet room. How was the masking of the sound done, by tuning fork or by his machine?

By placing the receiver to the ear in his machine, he does not get a pure air or bone conduction sound, but a combination of both, and a good deal of study is necessary to interpret these sounds.

DR. HAYS said that naturally most men were more interested in the practical application of this question than in anything else. The paper was so much along scientific lines that very few could really grasp much that had been said. All, however, were probably looking about for a new audiometer, and it would be interesting to know how the one spoken of by Dr. Fowler compares with others that have been made in the West, notably the instrument of Dr. Isaac Jones of Los Angeles. He claims to be able to make a differential diagnosis between conduction deafness and perception deafness. In regard to the audiograms shown, the question arises as to the value of these charts over the regular, ordinary hearing tests. Dr. Hays felt that this method of recording was far superior to the old method. During the past four weeks he has made some fifty audiograms which form an interesting study. It is questionable how much a diagnosis of nerve deafness means. Actually there should be little perception of sound of any kind. If we speak of cases of perception deafness and find on the audiometer that the patient hears the higher notes with great difficutly, what does that mean from a practical point of view? We speak of perception deafness, but we really do not know anything about it for in these cases, many

times, we manage to appreciably improve the hearing by common-sense treatment.

The most important point about Dr. Fowler's paper is the fact that we are getting to a practical way of measuring hearing. Whether we will be able to make differential diagnosis such as Dr. Jones claims to be able to do, remains to be seen, but when one can take hundreds of these cases and study them, he will find out something really definite and worth while.

DR. HAYS said that it was a great satisfaction to the patient to see his hearing accurately charted on an audiogram. And it also is a satisfaction to be able to work out percentages of hearing. Much as it means to us to make a differential diagnosis of a hearing defect, by actual experience we have found that what counts for most is the actual demonstration as to whether our methods of treatment have materially benefitter the patient. He stated that he had seen many cases of so-called nerve deafness which improved considerably as soon as the Eustachian tube received proper attention.

DR. BEAMAN DOUGLASS said he was very much interested in Dr. Wiegel's representation of the action of the Basilar membrane for tone, and would like to know whether Dr. Wegel knew of any laboratory instrument which would demonstrate this action for tone which would help us better to understand the physiology of the ear.

It seemed to him whenever he listened to an orchestra that the sound which entered the ear must be a composite tone and not an orchestral wave of many instruments playing different pitches, and that which reaches the ear must be something of the same kind of wave as one sees inscribed on a phonograph roll. After a phonograph roll is reproduced upon a revolving drum the form of identation on the roll can be studied and that which one sees is a single line which is a composite sound of all the different tones which, let us say, a quartet or even an orchestra is playing. No analysis of the phonograph identation enables us to separate these tones which we subsequently hear in our ears as tones from different instruments, that is to say, we are unable by phonographic analysis to separate the various instruments which are composing the tone inscribed on the phonograph. It is difficult to describe composite tone, but it seems to be our association that makes us translate this composite tone into various tones and pitches, and yet when listening to an orchestra one can subdue or subjugate certain pitches and hear only the one which he wishes, for instance, one can eliminate the sound of the strings and preserve only the sounds of the horn.

This made Dr. Douglass feel that the wave reaching the ear must be of a different quality from those that are inscribed on a phonographic roll, and yet the results upon hearing the orchestra and upon listening to a phonograph are exactly the same. According to Dr. Wegel's theory all the different pitches are introduced through the fibrous drum membrane and affect different parts of the cochlea of the basilar membrane.

The thing seems to be not entirely worked out as yet, for if every wave coming from an orchestra of which sometimes there are seventy-five instruments separately affects the drum of the ear and its distributing mechanism the membrane of the cochlea must be in a very delirious motion most of the time.

It has, therefore, always seemed to him that that which the ear receives was a composite tone and not at all the one which Dr. Wegel describes.

DR. WEGEL, replying to Dr. Douglass's question, said that at a meeting of the Otological Society in Atlantic City, Dr. Dench showed a model of the cochlea which he brought from some experimenter in England. This consisted of a brass container with small wires stretched across it along its length and covered with collodion so as to make a membrane with stretched transverse fibers. The whole was then filled with a fluid and a stapes and round window attached at one end. Small dust particles were placed on this membrane under the fluid and forks of differ-

ent pitches could be seen, by means of the orientation of the powder, to disturb the membrane at different places corresponding to the pitch.

I think the question of fixing of pitch is one of association. We have all been accustomed throughout our lives to hear various pitches which we have been taught to associate with some symbol, musical or otherwise and it seems to me that musical appreciation is simply a question of an association of the sounds we hear through the means of the stimulus of the tactile organ in the cochlea with the various associated circumstances which give pleasure or grief, etc. When orchestra music is played, practically all of the membrane is in action in a very complicated way and we have become able through a process of constant, though unconscious practice, to sit back, so to speak, and pay attention to the whole or to examine any detail of this complex vibration so as to pick out any single instrument and listen to it.

DR. KAHN said it seemed to him that Dr. Fowler had not taken into sufficient consideration the factor of sound conduction. The question of a given subject hearing better in a noisy atmosphere in contradistinction to their hearing in a quiet atmosphere. He thinks this is purely middle ear. That the bones in the middle ear are put into vibration first so that a lower sound following are carried across an already vibrating ossicular chain.

His impression was that the lower tones were produced at the near end of the cochlea. In catarrhal cases for example the lower tones were almost entirely affected. The idea of sounds bridging the basilar membrane therefore was not at all clear to him. Sound is produced not only by a continuous wave, but by a wave continually making and breaking. It is just as important for the membrane at the round window to vibrate back as it is for the stapes to start the wave. And then the Organ of Corti like any other scale comes into play. He could not get Dr. Fowler's basilar membrane idea fixed in his mind.

He could understand how sound could be masked up to a certain point on the scale, say, for example from the lower register up to a given point of the scale, but he could not understand how a certain island area, as it were, was left free and then the register beyond again masked.

DR. FOWLER said that the discussion opened up a large subject. If, however, Dr. Kahn would read the paper he would find that most of his questions were answered. These findings were definite and there was no question as to where the high and low tones were sensed. What do we know about air or bone conduction? We don't know much. Some men claim that all obstructive cases have also a nerve involvement. The main thing we have to go on to diagnose this is the diminished bone conduction; but it is perfectly conceivable that bone conduction may be diminished by other than nerve deafness. We have been using tests for our hearing and diagnoses the best of which are very crude after all. The tuning fork tests are definite as far as they go, but every tuning fork is different; we never strike it the same, it will vary as much as twenty to fifty seconds if we are honest and put down things honestly, and the same with bone conduction.

Increased bone conduction: If you examine bone conduction carefully you will be impressed by how few cases you find with increased bone conduction. I am getting more critical all the time about increased bone conduction.

Some of the points made by Dr. Gutman were well taken, but, as Mr. Wegel says, we have to take this in a tentative manner and try to get some basis from which to reason. In reading the paper, it was necessary to skip a great deal which clarifies this, but the measurements, as far as they go, are quite accurate and were made by men who are dependable. I simply saw that the patients tested responded accurately to the tests. There is always a difference in attention. a difference in education, as the Doctor said, but it has been found that musicians give no different data upon minimum detectible frequency change than do others.

Replying to Dr. Kahn's question as to his doubts regarding the hearing of high tones near the stapes end of the cochlea and of the low tones near the apex, Dr. Fowler said that this was proved years ago by Liebermann, by destroying different nerve areas of the basilar membrane by very loud tones. The lesions were found in stained sections to be situated as expected, proving that the high and low tones were sensed respectively at the upper and lower whorls of the cochlea. All laws of physics point to the same conclusion.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

April 23, 1924.

Papillary Carcinoma of Inferior Turbinate, Septum and Antrum. Dr. Saul Knopf.

DISCUSSION.

DR. SMITH said that these papillomatous conditions of a cancerous nature are rather unusual, and he recalled a case sent to Dr. Jonathan Wright some years ago which extended into the ethmoidal region and into the attic of the nose. The patient had had frequent hemorrhages and was later sent to Dr. Mosher of Boston. The ultimate result was that it extended into the brain, from which the patient died. Dr. Smith said he understood from Dr. Knopf that he thought this maxillary involvement was a mucocele, but he doubted this for the simple reason that any malignant tumor blocking off the antrum produces a secretion resembling that of a mucocele. Moreover, the X-ray in a mucocele shows a thinness of the bony wall so that it shows clearer than the X-ray in this case, which is rather opaque. Dr. Smith said he did not think there was any doubt but that this case was malignant, and of a more or less rare nature.

DR. HARRIS said he had just seen the case, and thought it was one that was so obscured by the recent use of radium that it was difficult to say how much of the necrotic reaction could be ascribed to the malignancy and how much to the radium. He was inclined to think that the radium was the cause, and hoped that with the disappearance of the radium reaction the condition would be more favorable. It was exceedingly active now, and the only treatment indicated was something soothing and quieting until this reaction was over.

DR. KNOPF expressed himself as feeling just as Dr. Harris did about the radium reaction. We really do not know the proper dosage of radium in the treatment of the various types of carcinoma, in view of the fact that some are more malignant than others, even of the same type. We will probably however learn more about radium, for we are on the right track, even as to the proper dosage.

Osteoma of Frontal Bone; Operation. Dr. E. Ross Faulkner.

This case was first seen Dec. 30, 1923, and the boy had then a large swelling extending a little to the right but occupying a fairly central position. The history was that he had fallen three months previously, and this had been slowly increasing in size since that time. At first he had some headache, but none later. An X-ray showed a very dense bone with a little frontal sinus on each side—a little more on the left than on the right—and extending into the ethmoid. Knowing that an ivory osteoma may occur in this situation and as this goes through both plates, there is no possible way of removing it except by exposing dura over the entire area, so I advised the patient to see a general surgeon, but the doctor, however, persuaded me to undertake it. So, after arming myself with an equipment of bone drills and sharp chisels, I undertook the case. I made the incision, as you see, through both eyebrows, crossing the spectacle line on the nose, etc., and getting an excellent exposure. Peeling off the soft tissue I found that the tumor projected outward, with a shelving process of the external plate of the frontal sinus lifted up on either side, giving it a sessile appearance.

I made a very good exposure, with a ronguer and went all around it; both frontal sinuses were full of pus and polyps, and this tumor was firmly attached to the inner plate. By inserting a pry under the edge of it I was able to lift it off from the inner plate, which was found intact. Then going down into the ethmoid, I found a mass of polyps, and a little extension of the osteoma in the ethmoid of the left side. Both ethmoids and frontal sinuses were cleaned out, etc. It was then packed with iodoform gauze, and the packing continued for four weeks. The deformity is much less than if I had made a primary union. As you see, the result is quite satisfactory. Dr. Faulkner said he was very glad he had undertaken the operation, for he thought a general surgeon would have had some difficulty in cleaning out the sinuses.

DISCUSSION.

DR. HARRIS said that Dr. Faulkner had a remarkably successful result; it was difficult to see how it could have been improved.

DR. MACKENTY thought the result very good indeed. Of course in children of that age one would not expect much deformity, since the frontal sinuses are not usually large. The method used at the Manhattan Clinic is very good—packing the flap underneath and endeavoring to get granulation tissue. Sometimes, however, it does not succeed and the wound has to be reopened, but if it can be kept clean it will relieve much of the deformity. Dr. MacKenty said he had had three of these cases in the past twenty years and he considered them difficult, especially if they involve the inner table; then they are dangerous. It was very fortunate that this case was operated by a nose and throat surgeon instead of a general surgeon, for the latter would never have been able to handle it.

Prolapse of the Ventricle of the Larynx. Dr. A. S. Wilson.

The patient came to the Manhattan Clinic, Dr. MacKenty's service, with a history of hoarseness and of having lost much weight in past six months. He had lost some twenty pounds and regained most of it. He stated that four years previously he had had a similar attack, which had not been so severe and had not losted as long. Examination showed very badly diseased tonsils, deviated septum, chronic ethmoiditis, and the teeth were not in good condition. Examination of the larynx showed a rounled tumor, apparently coming from both sides and anterior commisure and looking as if it was split in middle; cords not visible and the edges of split were ulcerated. Naturally the first thought was carcinoma. However, the man gave a history of having had a specific condition fifteen years before. He had had an ulceration of the throat, which had entirely destroyed the soft portion of the palate, and finally a portion of it fell off. He finally went to the Hot Springs of Virginia for treatment, and it was healed up. During the past summer he had a little cough, and his chest was examined but proved negative, and his sputum was negative. An X-ray of the chest showed some infiltration of both apices, and Dr. Law thought he probably had an active condition. His blood serum was normal, and his sputum was negative to tubercle bacilli. He had no pain in the throat, but simply complained of hoarseness. He was sent to Dr. James A. Miller to eliminate tuberculosis if possible. Dr. Miller thought the condition was not tuberculous, but that it looked more like carcinoma. An effort was made to eliminate every possibility before operating for carcinoma. The last suggestion was that it was a prolapse of the larynx following a syphilitic condition. The man has been a barker in country fairs, and had to use his voice a great deal. The suggestion of prolapse was made by a man who had observed such cases in Austrian officers, who had had a syphilitic history ten or fifteen years previously.

The patient was not given salvarsan on account of the condition in his chest, but he was given 120 grains of iodid of potassium per day, and the growth had diminished in size and the voice had improved, and

the whole appearance was improved. Two biopsies were made, and both reported inflammatory tissue.

Dr. Wilson said that it was impossible to say whether or not the man was going to get well, but that he did not know of anything else to do except to continue the iodid.

DISCUSSION.

DR. MACKENTY said he had been very much interested in this case. The first impression was that it was a malignant growth, but the point which militated against that opinion was an even condition which involved both sides of the larynx equally and did not seem to have a start in any one place, and there was no loss of motility. The fact that the patient had this double uniform condition involving both sides equally, was against it being tuberculous or malignant, but it was very difficult to make a diagnosis, though the one presented was probably correct. He had been growing suspicious of its being an inflammatory condition when a man from Vienna, who had had a great deal of experience among the soldiers and officers abroad, said he thought it was a typical case of prolapse of the ventricles. The case was an extremely instructive one, and it was very fortunate that it was not handled surgically. There is a probability of the condition becoming malignant eventually.

DR. HARRIS thought this was as interesting a case as had been presented during the evening. The history had a very important bearing on the diagnosis. Looking at it as seen tonight, he did not see how it was possible that such a diagnosis could be made. There was a very distinct bilateral condition and the whole larynx was inflamed. Everything else, however, had been eliminated. The Doctor's statement that the condition was very common among the Austrian officers was very interesting, too. As he remembered, these cases were very rare; he had not himself seen more than one or two in his life, and the picture in those was distinctly unilateral—limited to one side, the other side being normal. He had been wondering whether the Doctor in emphasizing the previous history had not touched the bottom of the matter, and whether there was not a specific origin in the matter—whether the case should be regarded as a pure prolapse of the ventricle. It seemed that the continuance of the iodid of potash was indicated, and he expressed the hope that Dr. Wilson would present the case again later.

DR. SMITH said he had seen a somewhat similar case of fibroma in the larynx in London a number of years ago which projected into the ventricle of the larynx just as this did. It was one of Sir Felix Semon's cases and he had removed it finally. Dr. Smith said he had seen only two cases of prolapse of the ventricle, and they were both unilateral, but that fact did not preclude a double prolapse. He did not, however, see why this condition might not be a syphiloma which was projecting into the ventricle and occluding the larynx. Entering externally and removing it might save the continuity of the larynx. He asked why Dr. Wilson had not given mercury with the potassium iodid. He seemed quite certain that it was not carcinoma.

DR. WILSON said he did not recall having said that the condition was common, for he did not know enough about them to make that statement, but this man from Vienna stated that he saw a goodly number of them during the war among the Austrian officers. The fact that it was bilateral was more suggestive than if it were unilateral. Mercury had been given at first, and then it was stopped because the man had some irritation of the bowels, and the K.I. was given instead. Dr. Wilson said he agreed that the diagnosis was not absolutely certain, but carcinoma and tuberculosis had both been eliminated and there remained only a syphilitic or parasyphilitic condition. He did not see how one could differentiate between those two conditions.

Argyrosis. Dr. J. D. Whitham.

The patient was a young woman, 28 years old, who stated that when 12 years old she had nasal diphtheria. Since then she has had nasal

obstruction and crusting, bad breath and cacosmia, somewhat relieved by nasal douching, which she has practiced for the past ten years. About four years ago, on the advice of a physician she began to instil each night a medicine dropper full of argyrol into each nostril. This did not seem to make her any worse or better. She had a considerable amount of stomach trouble, which had been diagnosed as visceroptosis. The entire mucous membrane of the nasal cavities was about the color of a slate. As she had an atrophic rhinitis, this could be very plainly seen. On the posterior pharynx it was almost quite black, though not so extreme as in the nose. Beneath the posterior pillar there was also a little tatooing from the pigment. It was interesting to speculate as to the reason this should have happened in the case of atrophic rhinitis when it might not have happened in a person with normal mucous membrane. The absence of ciliated epithelium in atrophic rhinitis and the lack of mucoid secretion prevented the removal of the argyrol granulas after the solution evaporated. The flattened epithelial cells probably were more permeable and so the pigment gained entrance into the subepithelial stroma. The atrophy of the lymphatics and blood vessels in the stroma probably prevented the migration of the pigment to other parts of the body.

DISCUSSION.

DR. HERZIG said he had never seen a case of argyrosis of the nasal membrane but had seen a number of cases of argyrosis of the conjunctival membrane. It was probably a matter of susceptibility in the individual.

DR. HARRIS referred to three cases presented before the Section in March where the discoloration was much more pronounced than in this one, so far as the external condition was concerned. In the discussion, it was brought out that probably argyrol is more dangerous than even silver, and someone made a very important point that the time had come when a warning note should be sounded against the use of argyrol by the laity, and that probably these cases were occurring more frequently than is recognized. One case was reported where the patient had been using argyrol as a mouth wash. It ought to be stopped except in proper cases.

A Clinical Study of Endocrine Disturbances in Rhinological and Laryngological Cases (Abstract). Dr. Louis Hubert.

Dr. L. Hubert discussed the possible role some of the glands of internal secretions may play in the causation of certain symptoms of the nose and throat. He considered briefly only such endocrine cases that he has seen at the Manhattan Eye, Ear and Throat Hospital. The symptoms the patients presented were grouped under the following headings:

I. Local symptoms.

1. Rhinological: a. rhinorrhea, b. sneezing.

2. Laryngological: a. choking sensation, b. hoarseness, c. irritation of pharynx (pharyngeal neuroses).

3. Respiratory: a. nasal obstruction, b. dyspnea, c. attacks of suffocation.

II. General symptoms and signs: nervousness, weakness, palpitation of the heart, mental excitability or mental dullness, headache, constipation or diarrhea; dry and pasty or moist and flushed skin, fine tremor or tongue and outstretched fingers, rapid or slow pulse, enlarged thyroid gland, certain changes in the distribution of the hair, certain abnormalities of the teeth, certain eye signs, as exophthalmos, dilatation of the pupils, etc., small sella turcica with erosion of the clinoid processes, enlarged thymus, etc.

The local symptoms were the dominant symptoms for which the patients came to the clinic. The general symptoms and signs were elicited on close questioning and careful examination, and helped to determine the underlying condition, i. e., what particular endocrine gland or glands were probably involved.

The rhinorrhea and sneezing cases were divided into three groups: 1. the mild hypothyroid, 2. the mild hyperthyroid, and 3. the indeterminate group. The hypothyroid cases were given thyroid extract gr. 1/10 t.i.d. The hyperthyroid cases, quinin hydrobromate gr. I to V t.i.d. The indeterminate cases responded to either thyroid extract or to quinin or to neither.

Dr. Hubert then called attention to patients who came to the clinic complaining of catarrh, or some vague throat condition, in whom close study revealed increased stimulation of the thyroid gland. Most of these patients characterized their discomfort in the throat as a choking sensation. The most constant and characteristic sign was an increased pulse rate, even at rest. The treatment of these patients consists in the localization and elimination of all foci of infection, by surgical or medical means, and by the administration of quinin hydrobromate. Those patients who cannot tolerate the quinin, adrenal residue, X-XV minims, t.i.d. is given. Hygienic and dietetic treatment must not be omitted.

Two groups of cases, belonging to the status thymicolymphaticus types were then discussed. In the one group there is nasal obstruction present and such patients are referred for removal of tonsils and adenoids. On further examination it is found that these patients in spite of their healthful appearance are very weak, and present certain signs that have been associated with a status hypoplasticus. The thymus gland may be slightly or not at all enlarged. In the second group of cases the thymus gland is enlarged and the most prominent symptoms are dyspnea and attacks of suffocation.

In commenting on the subject Dr. Hubert Smith said: Although we know very little of the conditions within the body that control the endocrine glands, the importance of studying the patients from the endocrine aspect, cannot be too much emphasized. If it does nothing else, it leads us to closer observations, and makes us better physicians. It forces us to consider the patient as a whole, and not only his upper respiratory tract. It makes us realize that many rhinological and laryngological phenomena can only be explained on the assumption that they are local manifestations of some general condition.

DISCUSSION.

DR. MACKENTY said he had seen all these cases and had seen the results. He had been somewhat skeptical, when these rhinorrhea cases came in, and Dr. Hubert put them under the treatment described, but could confirm what had been said.

DR. HERZIG asked what Dr. Hubert would substitute for local anesthesia; general anesthesia?

One hundred and fifteen of these cases have been tested for sensitization reactions; about 10 per cent gave positive reactions. The observations of Dr. Hubert upon the endocrine reactions of these cases has an important bearing upon the relationship of the general metabolism upon the development of allergic reaction because the allergic vasometer rhinitis cases are clinically identical with the endocrine cases. So there apparently must be some common ground in the etiological factor.

DR. GUTTMAN said he had been interested in this subject for a long time from the otological standpoint. We have to distinguish here the action of the endocrine disturbance from two standpoints: first, the mechanical effect; and, second, the intoxication effect caused by the endocrine disturbances. An enlarged thymus or thyroid presses mechanically upon the laryngeal nerve and will cause a laryngeal disturbance; the other disturbances caused by chemical dysfunction, is not so easily understood and there is much controversy about it. It is very difficult to say, for instance, a rhinitis nervosa which occurs at the time of the menopause. How much the menopause is the cause of the rhinitis.

To be Continued.